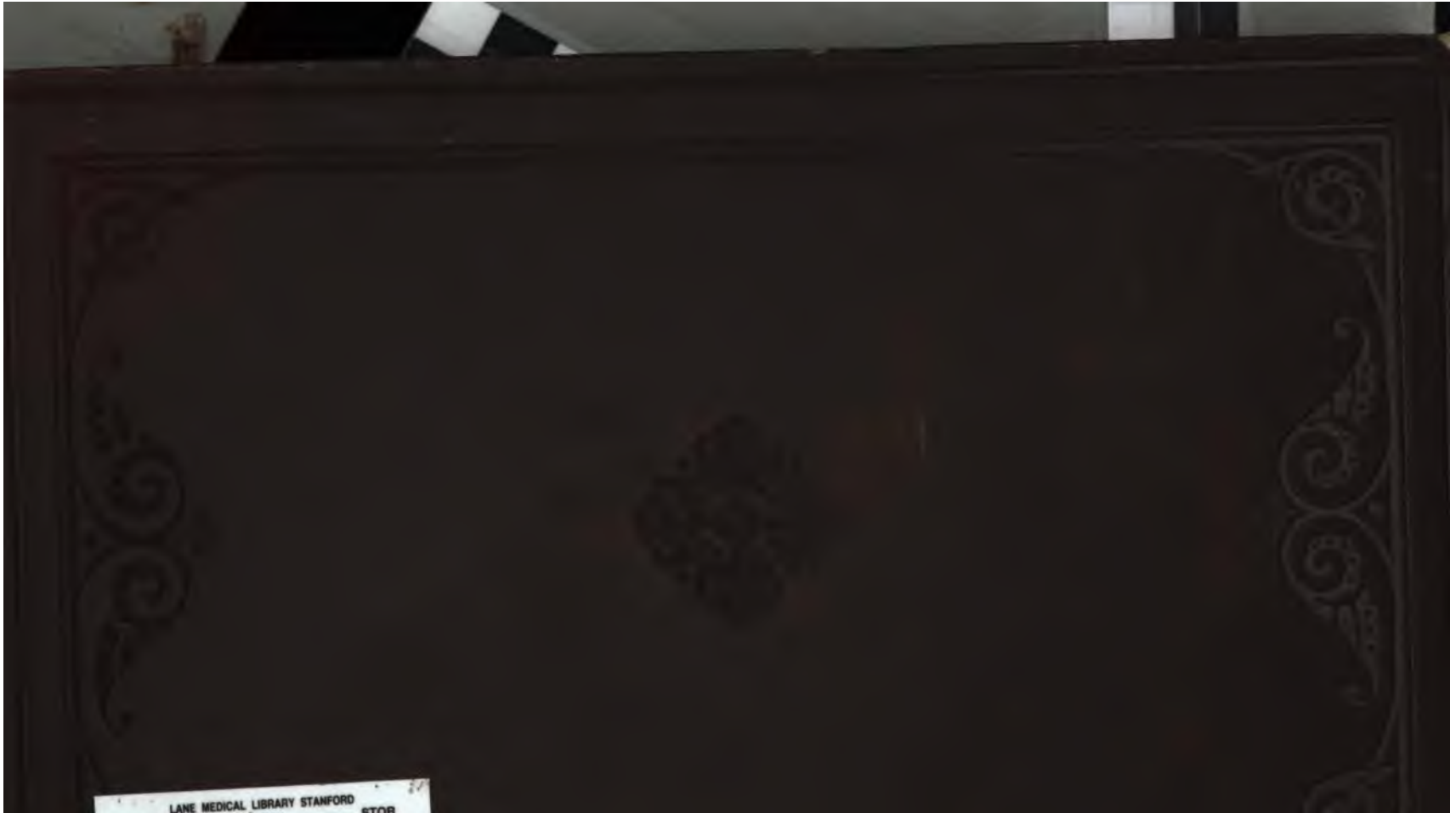

ON

ASTHMA:

ITS PATHOLOGY AND TREATMENT.

BY

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THIS IS INSCRIBED

IN ADMIRATION OF HIS RESEARCHES INTO THE NATURE OF

OBSCURE AFFECTIONS OF THE NERVOUS SYSTEM,

AND NOT THE LESS

IN GRATEFUL ACKNOWLEDGMENT OF MANY

OBLIGATIONS.

CONTENTS.

PREFACE.

THE intractability of asthma, which, some years ago, was thrust upon my notice on several occasions, induced me to make that disease the subject of a special study. I was the more tempted to do so by the conviction that there could be no prospect of satisfactorily coping with the complaint so long as, on the one hand, the "double capriciousness" was regarded as an essential element of its pathology; while, on the other, for the exhibition of remedies of the most opposite effects, there was no other indication but that they had been found useful in isolated instances. In the pursuit of that inquiry, little perspicacity was needed to discover that the prevailing obscurity on the nature and treatment of asthma, primarily and mainly, arose from the exclusive attention bestowed upon the dyspnœal paroxysms; whereas their constant antecedents and sequelæ, which form the life-history, as it were, of the disease, were either neglected or erroneously interpreted. It is readily intelligible, that a dyspnœa, which, apparently independent of organic lesions, suddenly arises and as suddenly subsides, cannot but be the subject of much speculation. A close examination, however, of all the facts connected with asthma, enabled me to conclude that this was merely one link in a chain of morbid processes, that com-

menced with a more or less insidious inflammation of the pulmonary tissue, and terminated with bronchiectasis and emphysema.

Such was the view that I formed of the nature of asthma, in my communication to the Medical Society of London in November 1873. Four years have since elapsed, and during that time the subject has continually engaged my attention. Ample opportunities for observation have strengthened the conviction, that the conclusion at which I then arrived is substantially correct.

The dependence of asthma on definite organic lesions, affords, on the principle that forewarned is forearmed, the best prospects, not only of averting the dyspnœal seizures, but also of arresting the final development of the disease. Neither of these objects was hitherto attainable, because the nature of asthma was supposed to consist of a mysterious derangement of the nervous system, and the exciting causes of the asthmatic paroxysm were generally regarded as inscrutable.

In the preparation of the following pages, I have consulted the writings of other observers, and have adopted their views when they appeared to be well founded. Where I have differed from generally received opinions, I have stated the reasons of my dissent. In the chapter on Treatment, I have confined myself to stating the principles upon which this may be advantageously conducted. Considering the multiform character of asthma and its differences in almost every single case, that plan seems to be preferable, as it saves the tedious enumeration of well known details.

71, *Wimpole Street*, W.,
May 1878.

"THE doctrine that certain so-called 'functional' diseases are constituted purely and alone by excess or insufficiency, altered speed, perverted rhythm, or modified association of the molecular changes *normally* attending structural acts, remains as yet mere matter of assumption."—W. H. WALSHE.

CHAPTER I.

INTRODUCTION.

THE so-called bronchial or spasmodic asthma is, to this day, perhaps the most obscure of all diseases. Notwithstanding the attention which asthma, on account of its frequent occurrence and the suffering it entails, has ever received at the hands of physicians, hitherto all their endeavours to elucidate the subject have only led to divergent opinions upon even the most essential points, and to the development of incongruous theories. Indeed, it is not saying too much that, among them there are not any two who fully agree in matters of either observation or inference; but all hold views at variance with one another, while each contends that only his own are consistent with facts, and in harmony with rational pathology.

Thus, amid controversy and much specious argumentation, the most important questions have remained unsettled. No understanding has as yet been arrived at respecting the essential characteristics by which asthma may be distinguished from any other dyspnœa. There are some who deny altogether its existence as a substantive disease;[1] others, on the contrary, regard as such every paroxysmal dyspnœa accompanied by sibilation, whatever else may be present at the time.[2] Others again object to the acceptation of the term in that wide sense in which it was used in the former systems of medicine; but although they greatly restrict the limits, they nevertheless differ in their definitions of the disease. According to Germain Sée,[3] asthma consists of three elements, namely, dyspnœa, bronchial exudation, and emphysema of the lungs. Again, German writers apply the term only to those dyspnœal paroxysms which cannot be referred by means of auscultation and percussion to structural changes of the thoracic organs. They maintain that, to warrant a diagnosis of pure bronchial asthma the bronchial mucous membrane should appear

[1] Beau, *Traité experimentale et clinique de l'Auscultation.* Paris, 1856, p. 132. — Traube, *Gesammelte Beiträge zur Pathologie u. Therapie.* Berlin, 1871. Bd. ii, p. 982.

[2] Hyde Salter, Clinical Lectures on Diseases of the Chest, *Lancet*, 1870. Vol. i, p. 147.

[3] *Asthme* in *Dict. de Med. et de Chirurg. pratiques,* tom. iii, 1865, p. 585.

healthy, nor should any other cause for the dyspnœa be discoverable.[1] Still, not all the supporters of this definition adhere to it throughout. They admit that asthma may be "complicated with" or "grafted on" other cardiac and pulmonary affections.[2] But, as these affections are themselves capable of producing asthmatic seizures, the distinction in a given case between this symptom and the supposed complication would be purely arbitrary.

Equally numerous are the theories that have been proposed to explain the nature of the disease. By some it is believed that a peculiar *materies morbi* attacks in a fitful manner at one time the skin, at another the joints, producing here gout or rheumatism, there cutaneous affections.[3] But when its energy is not exhausted by acting upon either of these organs, the "peccant" matter irritates the nerves of respiration and causes either coryza or dyspnœa. According to this view, asthma is merely the symptom of a constitutional disease, which manifests itself in the several ways just mentioned.[4] But the majority of writers, while admitting the existence of the supposed humoral

[1] Niemeyer, *A Text-Book of Practical Medicine.* 7th edition. London, 1870. Vol. i, p. 87.

[2] Lebert, *Klinik der Brustkrankheiten.* Tübingen, 1873. Vol. i, p. 443.

[3] Duclos, *Bulletin générale de Thérapeutique.* 15 Avril, 1861. Tom. lx, p. 299.

[4] Trousseau, *Clinique Médicale de l'Hôtel Dieu.* Paris, 1868. Tom. ii, p. 453.

disturbance, yet regard this as only an exciting cause.[1] They assume that the essence of the disease consists in a peculiar functional derangement of the nervous system, and more especially that portion of it which presides over respiration. The unanimity with which all, and even those whose opinions are deservedly the most esteemed, support this doctrine, would be almost sufficient to carry conviction as to its correctness. Still, although they agree in the main point, they widely differ in regard to the modes in which the nervous derangement shows itself. For there is not one muscle engaged in respiration that in its turn has not been supposed, by excess or insufficiency of its innervation, to be immediately concerned in the production of the asthmatic dyspnœa.

It would be tedious to refer in detail to the multitudinous causes assigned to the disease, and the conflicting statements of its symptoms. Enough has been said to show that the estimate formed of what is actually known of the subject is substantially correct. Indeed, the study of the literature unavoidably produces the impression that the asthma of to-day is still what it was at the earliest periods of the history of medicine —a generic term denoting paroxysmal dyspnœa. In this sense at any rate it is used by modern writers, whose elaborate classifications are intended to show

[1] Hyde Salter, *On Asthma*. 2nd edition. London, 1868, p. 14.

"the unity of the disease in its several forms".[1] And, like a true genus, asthma has thus its species and varieties, the number of which equals, if not exceeds those recounted in the classifications of Sauvage Cullen, and Richter.[2]

That asthma has hitherto so completely baffled every enquiry must be attributed to circumstances partly inherent in the disease, partly accidental. In the first place a certain allowance must be made for the peculiarity of its symptoms. The rapid transition from apparent health to imminent asphyxia, and the rapid return from this to a comparative or absolute freedom of respiration, are not only striking phenomena, but are unlike any produced by the recognised inflammatory affections, acute or chronic, of the thoracic organs. Moreover, physical signs, which in no other dyspnœa fail to show an adequate cause, seem of no avail in the present instance. Admitting, therefore, that the diagnosis of the disease is thus beset with unusual difficulties, still they might have been successfully met, if observation and the analysis of the symptoms had been conducted with the necessary precautions. Instead of this, the great majority of investigators could not free their minds from the prejudices and theories descended from

[1] Hyde Salter, *op. cit.*, p. 118.

[2] Wunderlich, *Handb. d. Pathol u. Therap.* 2nd Aufl., B. iii, p. 231.

the past. But, under the influence of these prejudices, all their observations and also their conclusions became necessarily coloured. Thus the theory that asthma is a nervous affection has almost uninterruptedly held sway since the time of Van Helmont, and it is, as above mentioned, the one adopted also by the vast majority of modern writers. There can be no doubt that they readily accepted it, without for a moment questioning its correctness. Indeed all their researches bear too evidently the stamp that they were commenced and throughout conducted with preconceived opinions. It was, in fact, their object to discover, *not what asthma is*, but, after prejudging the question, *how nervous asthma is produced*; and if any attempt was made to prove the assumed nervous origin of the disease, there is sufficient evidence that all reasonings proceeded from, and tended to, the same point. Hyde Salter found that the sonorous and sibilant rhonchi which accompany an asthmatic attack could not be referred to either an inflammatory swelling of the bronchial mucous membrane, or to a plug of mucus within, or to an organic stenosis of the air tubes.[1] Assuming that the râles could have no other organic cause, he therefore inferred that the lungs were healthy.[2] But because all diseases that cannot be traced to organic lesions produce their symptoms through the nervous system,

1 *Op. cit.* p. 36. 2 *Ibid.* p. 37.

he therefore concluded asthma to be a nervous affection.[1] Again, assuming that the physical signs were due to a spasm of the bronchial muscles—the possibility of which occurrence being far from ascertained—he argued that, as the phenomena are muscular, the primary disease is nervous.[2] It is superfluous to add that this is merely begging the question. Yet such is the foundation upon which Hyde Salter and many after him constructed their theories. And, though the imperfections of these were too obvious to escape notice, yet, to save the foregone conclusion, all the facts which, by no stretch of imagination, could be brought into conformity with it, were considered as "caprices". Now whatever asthma may be, it assuredly obeys definite laws, however unknown these may be in their essence. So that if the greatest number of facts cannot be satisfactorily explained, the seeming "double capriciousness"[3] belongs to the theory rather than to the disease.

Perhaps one motive was powerfully at work to render the enquiries less rigid than they ought to be. It so happens that almost all whose names are most intimately associated with the pathology of the disease had themselves been sufferers from asthma.[4] That they were anxious to arrive at true conceptions

[1] *Op. cit.*, p. 30.
[2] *Ibid.*, p. 50.
[3] *Ibid.*, p. 107.
[4] Floyer, Lefévre, Salter, Trousseau, etc.

of its nature there cannot be a moment's doubt. Still hope and fear must have so greatly influenced them that they could not have been unbiassed judges of their own cases. It is consoling, more especially to the physician, to know that the malady from which he suffers is due to a subtle nervous derangement, rather than to irreparable organic lesions. Considering, therefore, the comfort they would have derived from such knowledge, they were eager to be convinced by a show of reasons, which, had they not been actively interested in the matter, they would have rejected as inconclusive.

Nevertheless, the theory of the nervous origin of asthma has met, as above mentioned, with an almost general acceptance. Nor can this be surprising, considering the authority of those who have supported it, and the earnestness and conviction with which they have pleaded in its favour. Authority is still influential in medicine, and even such men as Biermer[1] think that "those who have written monographs on asthma *must* have known that this was a nervous affection". Another circumstance contributed to the same end, namely, that the theory, as it now stands, affords unusual facilities in the routine of practice. For, not only does the nervous system easily lend itself to all possible interpretations, but the teaching that parox-

[1] *Bronchial Asthma;* Volkmann's *Sammlung-klinischer Vorträge* No. 12, p. 3.

ysmal dyspnœa accompanied by sibilation is sufficiently characteristic of the disease,[1] supersedes the often difficult process of diagnosis; while in the failure of a well concerted plan of treatment it is consoling to know that the "capriciousness" of asthma baffles all calculation.

Such teaching, however, has led to the most serious consequences. The success and failure of the treatment are by no means always in direct proportion to, and the test of, the knowledge or ignorance of the pathological state which it is intended to relieve. Still it must appear inexplicable that a disease, which is supposed to have no palpable cause, and which neither kills nor even abbreviates life, should have hitherto resisted all remedial efforts. Yet so it is. Asthma is proverbially the most intractable of all diseases; and, so far as the results of the treatment are concerned, that belief is not unfounded. But it would be surprising if it were otherwise. The fact is, physicians are constrained to the humiliating confession that they have no principle upon which to base their treatment, but that in this they are guided only by the experience of the patients,[2] who are generally credited with an intimate acquaintance with the therapeutics of their case. The task of the physician is reduced to merely enumerating the remedies recom-

mended and to suggest the trial of one of these that might have escaped the attention of the patient. Such practice sufficiently explains how it is that the asthmatic has "to suffer many things from many physicians";[1] and it also accounts for the fact that the treatment of the disease is, at present, the lucrative pursuit of speculative druggists.

To expect an improvement of the treatment of asthma from the search after new remedies betrays a strange disregard to the lessons of its history. As matters are, even that discovery could only add fresh disappointment. Besides, it is admitted that none of those remedies hitherto in use have ever been exhibited under appropriate conditions, so that it would be rash to pronounce on their inefficiency without even a fair trial. But what is wanted is a better insight into the pathology of the disease, such as can be obtained only by the careful examination of ascertained facts. And whatever follows from these in logical sequence has at any rate a greater claim to correctness than a baseless theory, however useful this may seem in many respects. Then, doubtless, the knowledge of the pathology will lead to serviceable means of treatment, while it at the same time obviates exaggerated expectations and groundless despondency. The prospect of a radical cure may, indeed, not improve; but as the known nature of asthma can hardly fail to indicate the way

to check and prevent, such practically amounts to it. It was perhaps for that reason that the founder[1] of this branch of pathology congratulated himself on the discovery that many forms of asthma, considered to be of nervous origin, really depended upon emphysema of the lungs.

With the view, then, of establishing a more secure basis for the treatment of asthma, I now propose briefly to sketch its history, and to discuss the prevalent theory, before I proceed to show what I conceive to be its true pathology.

[1] Laennec, *Traité de l'Auscultation*, etc. 2nd edition, tome 1, p. 289.

ALL early historical traces of the affection at present called asthma are lost. Although the disease is said to be mentioned in the Bible, and described by Hippocrates, Aretæus, Galen, and Celsus,[1] there is not the least evidence that those remarks apply to the asthma of to-day. For in the former systems of medicine, all cases presenting the *same conspicuous* symptoms were, regardless of their anatomical differences, considered as of a kindred nature, and grouped into classes according to imaginary types. Thus *intense but intermittent dyspnœa* formed the typical characteristic of the class asthma; and this, with its numerous species and varieties, comprised a promiscuous collection of diseases having absolutely nothing in common but that one symptom. In that sense, however, the term asthma was employed until the commencement of the present century, so that even, at so late a period, empyema and dilatation of the heart were described as asthma paradoxon[2] and asthma nervo-

[1] Bergson, *Das krampfhafte Asthma der Erwachsenen.* Nordhausen, 1859, p. 6, *seq.*

[2] May. Hufeland's *Journal*, xix, 1804; quoted by Wunderlich, *op. cit.*, p. 233.

sum.[1] Moreover, as before the invention of auscultation and percussion, marked functional disturbances were the sole means of diagnosis, their close similarity in all diseases of the chest must have necessarily rendered an accurate distinction between the several known forms of them impossible.

It is only after percussion began to be practised that asthma can more clearly be discerned in history. As soon as certain cardiac affections, hitherto imperfectly or not at all known, were by means of it recognised during life, it became at once evident that the dyspnœal attacks which accompany them were not of nervous origin, as was until then supposed, but were the necessary consequence of the structural lesions. Corvisart,[2] who was the first to perceive this fact, was moreover led to infer that of the prevailing forms of nervous asthma, several, if not all, probably arose in an analogous way. But his means of diagnosis were as yet too limited to prove that supposition.

The task was thus reserved for Laennec.[3] His invention of auscultation brought physical diagnosis to such a degree of perfection as to enable him to determine the true value of the numerous forms of the "asthme des praticiens", and to range them in their proper places as mere symptoms of anatomically defined diseases. Yet in that sweeping measure he

[1] Heyfelder, Schmidt's *Jahrbücher*, Bd. viii.

[2] Laennec, *op. cit.*, p. 71.

[3] *Ibid.*, p. 78 *seq.*

could not comprise the intermittent attacks of dyspnœa, of which no adequate cause was discoverable by means of the stethoscope. In several such cases, which, however, he had occasion to examine only in the intervals between the attacks, he found the pathognomonic signs of emphysema and bronchitis. But though these affections sufficiently explain the dyspnœa, they do not explain its paroxysmal character. He was, therefore, at a loss how to account for this. What appeared to be an almost insurmountable difficulty was that there were no physical signs; and, as he valued these too highly to think that they could ever fail in any given case, he inferred that their absence denoted the absence also of anatomical changes. That inference seemed to agree with his speculations upon the nature of those cases. It was not likely that the trifling incidents reported as exciting causes of the attacks, such as sleeping in a dark room, could so rapidly produce anatomical changes in proportion to the intensity of the dyspnœa. Nor was it probable that if such were produced, they could as rapidly subside without leaving any traces. Here, therefore, was strong presumption for excluding structural changes from the asthmatic attacks. The following case, which soon came under his notice, seemed fully to confirm that opinion :—

"A man, 40 years of age, had suffered for a long time from a large ulcer of the left leg. The tissues of the

whole foot were greatly swollen, and of an almost stony hardness. The limb, with its dirty greyish colour, presented in many respects the appearance of elephantiasis. Usually there was abundant discharge from the ulcer. For the last five or six years the patient had been subject also to a slight cough, but never had dyspnœa or pain in the chest. By mistake he was placed in the medical wards, and remained there several days, but when about to be transferred into the surgical wards he was suddenly seized with extreme dyspnœa. It was noticed on that occasion that the discharge from the ulcer was much less than it had been before. The patient was sitting in a chair, and begged, in a state of inexpressible anxiety and with a gasping voice, to be relieved of the enormous weight which compressed his chest, threatening suffocation. The inspirations were short, very frequent, and at times convulsive. The pulse was rapid and thready. The chest had been frequently examined, but always with negative results. No cause of that formidable accident could be detected. The thorax gave everywhere the ordinary sonorous note on percussion ; only on the left side behind, over a space of a few inches, there was a slight deficiency of resonance. At that place also the respiratory murmur was feeble and accompanied by râles, whilst over the rest of the lungs air was heard freely to enter the air vesicles. The heart and large vessels seemed normal. Thus no clue whatever was obtained to the

nature of the dyspnœa, which, however, constantly increased in intensity. Treatment, consisting in bleeding, blistering, etc., was of no avail. The next morning the dyspnœa was so intense that the patient was in imminent danger of asphyxia. The symptoms somewhat resembled those of œdema of the glottis. The question was raised whether the dyspnœa might not proceed from laryngeal obstruction. Tracheotomy was proposed as the sole chance of preserving the patient's life. The operation was performed by Professor Roux, and it is hardly necessary to add that all due precautions were taken to ensure the free entrance and exit of air to and from the opening in the trachea. No amelioration, however, followed. The dyspnœa continued to increase, and the patient died the same evening. On examining the body it was found that the lungs were healthy and crepitating, with the exception of a small portion of the lower lobe of the left lung. That portion alone was in a state of hepatisation. In the bronchial mucous membrane there were small red spots. The heart and large vessels were normal. No pathological changes could be detected in the head and abdomen. The subcutaneous tissue of the leg had undergone lardaceous thickening, and the tibia was necrosed."[1]

There is little doubt that the dyspnœa in this case

[1] Andral, *Clinique Médicale*, tome i, p. 250. Paris, 1829; quoted by Laennec.

was produced by *embolism of the pulmonary artery.* The protracted disease of the leg favoured the formation of a thrombus, and the patient's removal from one ward into the other probably caused the disastrous impaction of the plug. Laennec, however, was ignorant of that pathological process. His attention was wholly engrossed by the changes in the pulmonary tissue, which, however, were apparently too insignificant to explain the dyspnœa and death. As it was, the case seemed, on the one hand, to confirm his inference that the absence of physical signs was conclusive of the absence also of organic lesions; on the other, to show that without these, there may be intense and even fatal dyspnœa. Thus the pathology of the case remained obscure, and with it the nature of similar paroxysms. In that difficulty the traditional theory of a pulmonary spasm suggested itself as a ready means of solving the question. From the researches of Reisseisen, the bronchi were known to be provided with muscular fibres, whose function, however, was as yet secret. Still, from analogy, Laennec assumed that they, being muscles, were endowed with contractility, and therefore liable to spasm. Such a spasm, he thought, might probably have been the cause of that fatal dyspnœa; but whether this supposition were correct or not, the case itself seemed to indicate the existence of a nervous asthma. Yet, notwithstanding the negative results of physical dia-

gnosis and *post mortem* examination, he had doubts on the subject. Hesitatingly he admitted spasmodic asthma, with this limitation, however, that it generally consisted of nervous and organic affections combined.

For a long time Andral entertained similar doubts as to the admissibility of a nervous asthma. Experience had taught him how deceptive it was to exclude, on the ground of the negative results of physical diagnosis and *post mortem* examinations, the possible presence of pathological changes.

"A dock labourer, fifty-three years of age, was admitted into the Charité Hospital suffering from acute articular rheumatism. For about two months he had also been subject to a severe cough. The expectoration was very thick and tenacious. On percussion, repeated several times, the chest always gave a full and sonorous note. On auscultation, there was distinct vesicular respiration over the whole left side, but over the upper and the middle lobes of the right side mucous râles were heard. There was no dyspnœa. The treatment consisted in several venesections, on account of the inflammation of the joints. One day, during a severe fit of coughing, the patient was suddenly seized with a violent attack of dyspnœa. The orthopnœa and severe fits of coughing persisted during the rest of the day and throughout the whole night. The next morning asphyxia was imminent. The face was swollen and cyanotic; the extremities

were livid. No pulse could be felt at the wrist. The patient pronounced with difficulty and with a gasping voice only a few words, requesting to be relieved of the enormous weight which he said he felt in the right mammary region, and which threatened to strangle him. The resonance of the chest had not diminished. Respiration was puerile over the whole left side; at the right base behind, mucous râles were heard over several spots. On the right side, also from the clavicle to below the nipple, and in the supra-spinous fossa, there were neither the normal respiratory murmur nor râles, although the chest was forcibly expanded. The probable diagnosis at the time appeared to be emphysema of the lungs, but scarcely was the examination finished, when the patient died.

"On *post mortem* inspection it was found that, with the exception of a general venous congestion, there was nothing abnormal in the cranial and abdominal cavities. The heart was of normal size, and the right side contained black and almost coagulated blood. The lungs were gorged with blood, as they are in a great many bodies, but nevertheless buoyant when thrown into water. There was nothing abnormal in the larynx and in the trachea. We thus remained in complete ignorance as to the causes of the symptoms and death. We could not find anything to explain the absence of the respiratory murmur, which we had noticed during life, in the upper lobe of the right lung, and which we had attributed to

an emphysema. But on slitting the bronchi, the blunt point of the scissors encountered at the origin of one of the larger air-tubes a mass of thick, almost solid, mucus, which had stopped up the membranous canal like a cork. It was gradually thinned out and prolonged into the interior of the bronchus. The mucous membrane was very red."

"We may be astonished to find", Andral proceeds, "that in this case such serious symptoms had resulted from the obstruction of so small a portion of the lungs, while in a large number of patients with such advanced disease of the organ as to render this almost unfit for respiration, life is yet sustained for a long time, and there is frequently not even much dyspnœa. The reason is, that here the reduction of respiratory surface is gradually diminished, while in our patient the obstruction was sudden."

"*Let me also remark that, if in this case the inspection of the lungs had not been made with care, one might have been led, in the absence of appreciable lesions, to regard it as one of nervous asthma, and the dyspnœa, which wholly depended upon a mechanical cause, would have been considered as a remarkable instance of a metastasis of rheumatism to the lungs.*"[1]

That valuable lesson, however, was forgotten as soon as the case previously mentioned[2] came under his

[1] Andral, *Clinique Médicale*, tom. i, p. 215. Paris, 1829.

[2] *Vide* p. 16.

observation. Here no anatomical changes sufficient to explain its pathology could be detected by the most careful examination, and Andral, like Laennec, therefore concluded that none existed. Although, as already stated, the dyspnœa was really produced by embolism of the pulmonary artery, Andral nevertheless thought the evidence so convincing as to remove at once all possible doubt. "If there is a case", he exclaimed, "that may properly be called nervous asthma, it is assuredly this."

The origin of an asthmatic seizure Andral attributed to a spasm of the bronchial muscles, and this again to an irritation of the pneumogastric and phrenic nerves. In many cases of extreme dyspnœa with intolerance of the recumbent posture, in which the lungs were to all appearance in a perfectly normal condition, he found enlarged bronchial glands pressing upon those nerves, and therefore asserted that such irritation may be a frequent, if not the constant, cause of asthma. But, although his observations are correct, his inference is not so. It frequently occurs that the pneumogastric is firmly embedded in enlarged bronchial glands, without necessarily entailing dyspnœa.[1]

Thus, upon the authority of Laennec and Andral, nervous asthma was admitted as a substantive disease

[1] Valentin, *Versuch einer physiol.-Pathologie der Nerven*, Leipzig, 1864. Abth. ii, p. 122.—Budd and Holmes, quoted by Lebert, *op. cit.*, [illegible], pp. 654 and 658.

into the present nosology. But the study of pathological anatomy, which had already begun to be zealously pursued, was not altogether favourable to that view of the matter. There was the tendency to connect the symptoms observed during life with the results of *post mortem* examinations, and to look with a certain distrust upon all such cases in which that connection was not established. Many dissentient voices were, therefore, raised against the supposed independence of asthma on structural causes. Rostan,[1] more especially, urged that in the numerous instances of it which had come under his notice he had always found pathological changes sufficient to account for the dyspnœa; and saw, therefore, no reason to assign this to a derangement of the nervous system. He appears, however, to have employed the term asthma in a manner too vague to give weight to his objections. So that the demonstration of the symptomatic nature of the disease in his own cases does not necessarily apply to all others.

The views which Louis[2] adopted of the pathology of emphysema considerably influenced also the views of physicians as to the nature of asthma. Although he studiously avoided even to mention the term, he never-

[1] "Mémoire sur cette question; l'asthme des vieillards, est-il une affection nerveuse?"—*Nouveau Journal de Médecine, etc.*, Sept. 1818.

[2] *Emphysème*, Diction. en 25 vol., tom. xi.

theless plainly indicated that he regarded not only both affections as identical, but also the dyspnœal attacks, which Laennec was unable to account for, as the earliest symptoms of emphysema. With Louis, emphysema was not an accidental complication of bronchitis, nor due to a disturbance of the inspiratory traction. With him, it was, on the contrary, a substantive disease, consisting of a peculiar nutritive derangement of the pulmonary tissue, and often distinctly traceable to a hereditary predisposition. Pre-eminently chronic in its course, it commences during infancy, and gradually reaches its complete development in the prime of life. But the morbid changes which the organ undergoes betray themselves during their progress by no *objective* phenomena. Their existence is revealed only by dyspnœal paroxysms, which, if dating from childhood, are almost pathognomonic of the disease.[1]

Lefevre,[2] however, reverted to the opinion of Laennec. Himself an asthmatic, he had watched his own case and that of a friend, and arrived at the conclusion that the dyspnœa could be produced only by a bronchial spasm. In looking thus upon asthma as a nervous affection, he still was not prepared to deny the possible existence of organic lesions; adding, however, that if such were present, they were of so fleeting a nature as

[1] *Loc. cit.*, p. 347.

[2] "Recherches sur l'Asthme, Mémoire couronné par la Societé de Médecine de Toulouse."—*Journal Hebdomad.*, 1835.

to elude detection. The essence of the disease he considered to be an increased irritability of the nerves of the lungs, in consequence of which the slightest irritation applied to the bronchial surface induced spasm of the bronchial tubes. The cases, however, which he himself reported, do not confirm his views. It is difficult to recognise a nervous affection, much less a bronchial spasm, in the dyspnœal attacks, which terminated always with the expectoration of thick masses of mucus. The expectoration was of a grey colour, very viscid, in consistency like boiled maccaroni, filamentous in shape, and, when disentangled, appeared as if moulded to the bronchi. Sputa of that kind sufficiently distinguish the preceding dyspnœa as the symptom of a fibrinous (croupous) bronchitis, and there can be no doubt that Lefevre and his friend were subject to that disease.

Although hitherto a spasm of the bronchial tubes had been confidently asserted to be the immediate cause of an asthmatic seizure, there was as yet no direct evidence even of their contractility. The experiments of Prochaska, Reisseisen, Haller, Varnier, Treviranus, and Krimer,[1] were not sufficiently conclusive in that respect. Some of these observers had only noticed that the collapse of the lungs on opening the thorax of a living animal was more complete than on opening that of a dead one, and attributed it to the combined

[1] Henle, "Uber Tonus, Krampf u. Lähmung der Bromhien u. über Expectoration."—*Zeitschrift f. rat. Mediz.*, Bd. i, 1844.

effect of the vital contractility and the elasticity of the organ. Others produced contraction of the air-tubes by the application of concentrated acids, opium, extract of belladonna, and by mechanical irritation. Others, again, failed in these experiments. And, lastly, Wedemeyer found that, whilst upon mechanical and electrical irritation the trachea, the larger bronchi and their ultimate ramifications exhibited no sign of contractility, the intermediate tubes of from one-third of a line to one line in diameter, gradually narrowed to an almost complete occlusion.

Budd[1] repeated these experiments, but failed to obtain the same results. He, therefore, rejected the theory of a bronchial spasm, and even doubted whether the circular fibres were muscular, as alleged. But although he regarded most cases of asthma as merely the mistaken dyspnœa of emphysema, still he admitted that there were some which, by their seeming independence of structural lesions, constitute a peculiar form of disease. Of their pathology, however, he could supply no precise information, but only the suggestion that in all probability morbid changes in the blood induce asthmatic attacks by spasm of the diaphragm and other muscles of respiration.

As the bronchial contractility was denied as often as asserted, Dr. C. J. B. Williams[2] again took the question

[1] *Medic.-Chir. Trans.*, vol. xxiii, 1840, p. 37.

[2] *The Patholog. and Diagn. of Diseases of the Chest.* 4th edition. London, 1840, p. 320 *seq.*

up in order to settle the matter in dispute. He, indeed, succeeded in showing, by experiments conducted with all necessary precautions, that mechanical, as well as chemical and electrical stimuli do produce contraction of the air-tubes. Thus the theory of a bronchial spasm obtained the support of experimental physiology. And even those who until then wavered in their opinions as to the possibility of such a spasm saw now no reason for doubting, but readily accepted that doctrine. Dr. Williams himself, assuming a *plus* and *minus* state of contractility, described two forms of asthma—the one spasmodic, the other paralytic. The former was supposed to be represented by the isolated attacks of dyspnœa which generally accompany emphysema; the latter by those which appear in connection with chronic pituitous catarrh.

Yet one link was wanted to complete the chain of evidence in favour of a bronchial spasm. As yet nothing was known of the innervation of the bronchi. Irritation of the vagus had produced no effect upon their calibres, and Dr. Williams was therefore led to conclude that "the muscular fibres seem not to be excitable through the nerves of the lungs".[1]

Soon, however, Longet[2] not only confirmed in the main the results obtained by Williams, but added that in

[1] *Op. cit.*, p. 331.

[2] *Traité d'Anatom. et de Physiol. du Syst. Nerv.* Paris, 1842. Tom. ii. p. 289.

his experiments irritation of the pneumogastric nerve always produced spasmodic contraction of the bronchi, whereas section of the nerve led to emphysema. Of the bronchial stricture he could satisfy himself by watching with a magnifying glass, but as to the occurrence of a similar process in the pulmonary tissue he had no means of judging. Still, he believed that such took place, although the presence of muscular fibres in the air-vesicles was then as doubtful as it is to-day. For, since section of the vagus causes emphysema, *i.e.*, distension of the air vesicles, there must be muscular fibres which, if liable to paralysis, are liable also to spasm.

After many fruitless attempts, Volckmann[1] at last arrived at the same results. But he could not, like Longet, convince himself by direct inspection of the bronchial contractility and its dependence upon the vagus. He only inferred as much from the experiments, which he described as follows:

"In the trachea of a decapitated animal I placed a glass tube, drawn out at one end to a point, with a fine opening. Upon electrical irritation of the vagus, the flame of a candle held before that opening was deflected, and once even extinguished. The same results were obtained when the thorax was opened, only the deflections of the flame were more feeble than before, because the collapsed lungs contain

[1] Rud. Wagner, *Handwörterb. d. Physiol.*, Bd. ii. Braunschweig, 1864, p. 586.

and expel on contraction a smaller volume of air than in their state of normal inflation. *The contractions of the lungs, however, take place in jerks.* Surely the lungs", he concluded, "would not be capable of such active movements unless these served some purpose in the economy of life."

These discoveries were at once utilised for the pathology of asthma. Upon the physiological data thus furnished, Romberg[1] described two affections of the vagus, namely, a bronchial spasm and paralysis, both of which are productive of typical dyspnœal attacks.

Bergson,[2] however, denied the existence of a paralytic asthma, because, according to the experiments of John Reid,[3] section of both vagi *produces no* dyspnœa so long as the animals are at rest and the supply of air unlimited—a fact duly confirmed by the later experiments of Rosenthal. Bergson, therefore, admitted only the spasmodic form of the disease, consisting in paroxysmal constrictions of the *bronchi and air-vesicles*, in consequence of a morbid irritability of the vagus.[4]

It cannot fail to be observed that those hasty applications of the results of the experiment to the pathology of asthma led here, as they always do, to unwarrantable conclusions. If, indeed, Longet were correct in this,

[1] *Lehrbuch der Nervenkrankheiten.* Berlin, 1851.

[2] *Op. cit.*, p. 100.

[3] *Edinb. Surgic. and Medic. Journ.*, vol. li, 1839, pp. 286 and 273.

[4] *Op. cit.*, p. 105.

that bronchial stricture and emphysema are due, *the one to a plus, the other to a minus state* of irritability of the vagus, it would necessarily follow that, as asthma and emphysema are almost always associated, spasm and paralysis of the self-same muscles could co-exist at the same time, each independently of the other, manifesting itself in its own peculiar way. Such deduction necessarily follows from the premisses. As regards Volckmann, it is evident from the report of his experiments, as stated above, that he never saw the contractions of either the bronchi or the pulmonary tissue. The *jerking* movements to which he referred could not have been produced by the muscular fibres of the lungs. These muscles are unstriped, and their contractions are consequently slow and gradual. Such, indeed, had been particularly observed and reported by Williams,[1] and later by Wintrich[2] and Bert.[3] In fact, Volckmann had mistaken the contractions of the pectoral muscles, to which the electric current had travelled along the moist sheath of the nerve, for the inherent active forces of the lungs.

Notwithstanding the incongruity of the theory,

[1] "The contractions and relaxations are gradual and not sudden". *Op. cit.*, p. 330.

[2] "Krankheiten d. Respirationsorgane."—Virchow's *Handbuch d. Pathol. u. Therap.*, Bd. v, Abth. 1. Erlangen, 1854, p. 195.

[3] *Leçons sur la Physiol. compar. d. l. Respiration.* Paris, 1870, p. 375.

asthma was, nevertheless, almost unanimously considered to be a nervous affection, and more especially a bronchial spasm. Only George Hirsch[1] urged that, among a large number of cases that had come under his notice, he had never met with one in which the dyspnœal paroxysm seemed independent of structural lesions. If asthma were a nervous affection, its frequent occurrence in the male would be unintelligible, because functional derangements of the nervous system are almost exclusively met with in the female.

Benvenisti,[2] again, complained of the disregard to pathological anatomy in cases of asthma. There were numerous pathological processes, such, for instance, as embolism of the pulmonary artery, sufficient to produce the most intense dyspnœa, yet not discoverable by physical diagnosis. To conclude, therefore, from the presence of sonorous and sibilant rhonchi as to the presence of a bronchial spasm appeared to him to be unwarrantable.

Beau[3] and his pupil, Crozant,[4] regard asthma as a chronic bronchial catarrh, accompanied with very viscid secretion. Such sputa, they maintain, are capable of obstructing the bronchi, and of thus producing the dyspnœal attacks, as well as the sonorous and

[1] *Spinal Irritation.* Königsberg, 1843, p. 248.
[2] *Commentario sull' asma.* Padova, 1859.
[3] *Op. cit.*, p. 132 *seq.*
[4] *De l'Asthme.* Paris, 1851.

sibilant rhonchi—their "râles vibrants". With the displacement of the mucous plug into a larger bronchus, or on its expulsion by means of a fit of coughing, the dyspnœa ceases, and with it also the râles disappear. This form of bronchitis is, in their opinion, due partly to an idiosyncrasy of the patient, partly to exciting causes, which greatly vary in different individuals.

The views of Beau, however, met with but little support. Apart from their inherent imperfections, the belief in the nervous origin of the disease was too deeply rooted to be readily abandoned upon the mere denial of its foundation. Beau omitted to offer any proofs that a bronchial spasm was impossible or improbable. He declared the experiments of Williams and Longet as not sufficiently convincing, and therefore endeavoured to substitute his theory for theirs.

But amongst the physicians who formed their opinions mainly from observations in hospitals and from the study of pathological anatomy, some sided with Beau, so far, at least, as regards the non-essential nature of the disease: others considered the matter as still an open question. Amongst the latter Canstatt[1] and Wunderlich[2] may be mentioned, on account of the reputation which their works enjoyed and the influence they consequently had upon many practitioners. Both thought that, in cases in which

[1] *Handbuch d. Med. Klinik.* Erlangen, 1855., Bd. ii, p. 698 *seq.*

[2] *Op. cit.*

the repeated and careful examination of the chest failed to show the presence of organic lesions, the dyspnœa might be attributed to a derangement of innervation, and, more especially, to a bronchial spasm. They added, however, that the dyspnœal attacks of presumedly nervous origin were, after but a short persistence, invariably accompanied by emphysema of the lungs. And as, according to the teaching of Louis, emphysema was a disease of gradual and insidious development, it therefore seemed to them highly probable that the asthmatic paroxysms were merely its precursory symptoms. The question thus remained unsettled. Moreover, Canstatt held that an accurate diagnosis was a matter of not much importance, because, whatever may be the relationship between asthma and emphysema, anti-spasmodics were equally beneficial in either case.

Traube and Villemin alone denied the nervous origin of asthma. A fluxionary hyperæmia of the bronchial mucous membrane was, according to Traube,[1] the sole cause of the dyspnœal attacks. He attributed its occurrence in only a limited number of persons to a certain predisposition on the part of their respiratory organs. The predisposition probably consisted in a deficiency of the elastic tissue of the lungs which allowed of a ready distension of the blood-vessels. So that a trifling cause, the effect of which is not perceived by the healthy, would

1 *Loc. cit.*

in individuals so disposed produce a swelling of the bronchial mucous membrane. That pathological condition, however, was not accessible to diagnosis during life, and might elude detection even on *post mortem* examination. Virchow[1] suggested that the elastic tissue of the lungs probably underwent a process of softening and absorption, somewhat resembling osteomalacia. Be this as it may, Traube had often observed that, after inflation of the lungs, some lobules did not collapse so completely as others, although the bronchial tubes that led to them were pervious. Moreover, the antecedents and sequelæ of each case forced upon him the conviction that certain nutritive changes existed in the lungs. Admitted this much, it was easy to account for an asthmatic paroxysm. The occlusion of the bronchi by the swelling of their mucous membrane caused an accumulation of carbonic acid in the blood, which by its impurity irritated the centre of respiration, inducing tetanic contractions of the diaphragm and the auxiliary muscles of respiration.

Villemin[2] professed to have demonstrated the pathological changes that deprived the pulmonary tissue of its elasticity, and predisposed the bronchial mucous membrane to hyperæmia. He stated that emphysema originated in a proliferation of the inter-capillary nuclei, whose advancing growth tended to compress the alveo-

[1] *Deutsche Klinik*, 1860, p. 463.

[2] *Archives Générales*, 1866, p. 580.

lar vessels. As the nutrition of the air-vesicles became impaired, they were unable to efficiently perform expiration. At the same time, the respiratory surface was reduced, and the blood accumulated in the bronchi to such extent as to convert their mucous membrane into a kind of erectile tissue. This condition gave rise to no symptoms, either subjective or objective. Its existence became manifest only by the readiness with which trifling incidents produced their effect. Hyperæmia rapidly ensued, and led to the dyspnœal attacks that other writers consider as nervous asthma. The chronic inflammation of the alveoli, described by Villemin, is, however, not recognised by other observers.[1]

In the meantime a new theory was brought forward. Wintrich[2] had again inquired into the bronchial contractility; and, after many unsuccessful attempts, at last succeeded in satisfying himself of its existence. Nevertheless, he concluded that a bronchial spasm was impossible, both upon physiological and clinical grounds. That cases occurred, in which the dyspnœa was due to a primary disturbance of innervation, he had frequent occasion to observe. But for their symptoms it was possible to account only by assuming, as Budd had suggested long before, a tetanus of the

[1] Lebert, *op. cit.*, Bd. i, p. 402.—Cornil and Ranvier, *Manuel d'Histolog. patholog.* Paris, 1876, p. 688.

[2] Virchow's *Handbuch der spez. Pathol. u. Therap.*, Bd. v, Abth. 1. Erlangen, 1854, p. 190-201.

diaphragm and other muscles of respiration, accompanied, perhaps, by a spasm of the glottis. Wintrich was, however, not prepared to deny altogether the participation of the bronchial muscles in the process, but he thought that, if such took place, their part in it would be only subordinate.

Soon the results of experimental physiology seemed to support the theory of Wintrich. Duchenne[1] demonstrated that faradisation of the phrenic nerve caused tetanus of the diaphragm. In the animals experimented upon, that operation was followed by distension of the lower half of the thorax, projection of the epigastrium, and extreme dyspnœa, the frequency of respiration being at the same time considerably diminished. Death, however, invariably followed when the electrical irritation of the nerve was continued for *more than a few minutes.* Unacquainted with the opinions of Budd and Wintrich, Duchenne was at first inclined to attach no importance to his discovery, because he thought a tetanus of the diaphragm was not as yet known to occur in man. Vallette, however, subsequently met with and reported a case of acute articular rheumatism, in which a fatal attack of dyspnœa had lasted *for several days uninterruptedly,* and from the symptoms of which he distinctly recognised a tetanus of the diaphragm. Duchenne[2] thereupon concluded

[1] *De l'Electrisation localisée.* Paris, 1855, p. 459.

[2] *Op. cit.* 2nd edition, 1861, p. 917.

that a tonic spasm of this muscle was the main cause of the asthmatic paroxysm.

Additional evidence in support of this view was subsequently supplied by Bamberger.[1] In a case of fatal dyspnœa he had occasion to observe the tetanus of the diaphragm, and to convince himself by *post mortem* examination, of the absence also of structural lesions. Although the symptoms of that case by no means resembled those usually assigned to asthma, he, nevertheless, regarded them as characteristic of the disease. Reviewing then the theories held on the nature of the affection, he arrived at the conclusion that the presence of the bronchial muscles did not warrant the assumption of a bronchial spasm. If this existed, he would expect to find the diaphragm ascended, the intercostal spaces drawn in, and a dull resonance on percussion; indeed all the physical signs described by Williams and Bergson in conformity with their theory. But, as yet, he had never met with a case presenting those symptoms. So far as his observations went, he had always found dilatation of the thorax and hyper-resonance on percussion. These signs could be accounted for only by assuming a tonic spasm of the diaphragm and the auxiliary muscles of respiration; and he therefore inclined to the opinion of Wintrich, Duchenne, and

[1] *Ueber Asthma nervosum: Würzb. Mediz. Zeitsch.*, Bd. vi, 1868, p. 102-116.

Valette. He divided, however, asthma into a central and a peripheral or reflex, the latter generally accompanying emphysema, bronchitis, pleuritic effusion, and tuberculosis. The tonic spasm of those muscles was the most frequent occurrence, but he had noticed also a peculiar form of asthma, especially in hysterical females. This consisted in clonic contractions of the respiratory muscles, producing a distinct *inspiratory* dyspnœa; and although this was generally supposed to be pathognomonic of laryngeal obstruction, the glottis was found pervious in the cases he had observed. Moreover, he held that paralysis of the diaphragm might also be a cause of asthma.

Lehmann,[1] Kidd,[2] and Sée[3] advocated similar views concerning the mechanism of the asthmatic dyspnœa. But while the former considered this as a mere symptom, Sée insisted that asthma was always a substantive disease, accompanied, but never produced by organic lesions. He recognised, however, only one form of it. In his opinion, asthma was essentially a neurosis of the vagus and its branches, and manifested itself by a tetanus of *all the respiratory muscles*, and by a bronchial exudation. The presence of these two "elements" combined was alone characteristic of the affection, whereas emphysema of the lungs was only

[1] On Pathogenesen of Asthma, Bibl. for Laeger, 1866.—Virchow's *Jahresb.*, 1866, vol. ii, p. 103.

[2] *Dublin Quarterly Journal of Medical Science*, 1861.

[3] *Loc. cit.*, p. 583 *seq.*

their constant attendant. Emphysema, he maintained, was not an essential, but merely the consequence of the inspiratory traction during, and the bronchial exudation after, the attack.

A detailed examination of this theory would lead too far. Whatever may be thought of the primary derangement of the motor innervation, there is, at any rate, no evidence as yet that the bronchial exudation proceeds, as alleged by Sée, from an exhaustion of the vagus. Schiff,[1] indeed, and recently Genzmer,[2] maintained that section of the vagus was followed by nutritive changes of the pulmonary tissue ; but Traube[3] and others have shown that the supposed vaso-motor disturbances are really produced by the entrance of foreign substances into the lung. Sée has then failed to account for one of the essential elements of asthma, and for the not less important relationship between this and emphysema.

The innervation of the lungs was, meanwhile, again the subject of inquiry. Knaut[4] was enabled fully to confirm the statements of Longet and Volkmann, that irritation of the peripheral portion of the vagus produced contraction of the lungs. Loewinsohn obtained

[1] *Die Ursache d. Lungenveränd. n. Durchschn. d. pneumogast. Nerven. Arch. f. physiol. Heilkunde*, 1847 and 1850.

[2] Gründe für d. pathol. Veränderungen d. Lung.—Pflüger's *Arch.*, Bd. viii.

[3] *Loc. cit.*, p. i, 113 and 132.

[4] *De vitali, quæ dicitur, pulmonum contractilitate, nervis vagis irritatis*, Diss. inaug. Dorp., 1859, p. 33 *seq.*

very different results from his experiments, and he was led to conclude that the pneumogastric was an inhibitory nerve of the bronchial muscles, so that only its section or paralysis was followed by their spasmodic contraction.[1] Rosenthal[2] did not succeed in verifying either of these views. In all his experiments, irritation of the central or peripheral portion of the vagus had no effect whatever upon the movements and volume of the lungs when the thorax was opened. He, therefore, doubted whether the vagus contained centrifugal fibres, that had any influence upon the respiratory movements. These conflicting opinions, however, were soon reconciled by Rügenberg.[3] He found that the lungs were influenced by the contraction of the œsophagus, to which they are attached by means of loose connective tissue. When, on irritation of the vagus the œsophagus contracts, the stomach, and with it the diaphragm, is drawn upwards, and the lungs thereby compressed. Hence the oscillations of the manometer, which the physiologists abovementioned had observed in their experiments, and

[1] *Experimenta de nervi vagi in respirationem vi et effectu*, Diss. inaug. Dorp., 1858, p. 41 *et seq.*

[2] *Die Athembeweg. u. ihre Beziehung. z. n. vagus.* Berlin, 1862, p. 232.

[3] *Ueber d. angeblich. Einfluss d. n. vagi auf die glatten Muskelfasern d. Lungen.*—Studien d. Physiol. Instit. z. Breslau 2 Heft. Leipzig, 1863.

erroneously attributed to bronchial contractions. For when the lungs were completely separated from the surrounding structures, they remained immovable on irritation of the vagus.

Regardless of the unsettled condition in which the question as to the innervation of the lungs thus remained, Hyde Salter, Duclos, and Trousseau nevertheless attributed asthma to a bronchial spasm. The essence of the disease was, according to Hyde Salter, a peculiar affection of the nervous system in general, and more especially that portion of it which presides over respiration. He held that the morbid irritability was mainly localised in the circular fibres of the air-tubes. Designed to guard the alveolar portion of the lungs against the contact with foreign bodies, these muscles are said to contract, in health, upon the application of various stimuli.[1] But asthma mainly consisted in this, that the contractile elements were readily disposed to take on spasmodic action, from causes to which they would hardly respond under normal conditions. It appears, however, that the function with which Hyde Salter endowed the bronchial muscles is, even in health, but imperfectly performed by them. As is well known, foreign substances easily penetrate to the alveoli.

Duclos, and again Trousseau, assigned the origin of asthma to humoral disturbances. Duclos[2] observed *two* cases in which, several years previous to the appear-

ance of asthma, the patients had suffered from cutaneous affections; and he therefore inferred, as Bouilland did, that a herpetic principle in the blood produced eczema and psoriasis upon the bronchial mucous membrane, and thus caused a spasmodic contraction of the air tubes. Trousseau enlarged still more upon that view. With him, asthma was a neurosis dependent on a peculiar diathesis. The same morbid changes in the blood that gave rise to gout, rheumatism, and hæmorrhoids, led to spasm of the bronchi, when the "peccant" matter was eliminated by this channel.[1]

The theory of Dr. J. Burdon Sanderson[2] differs from all the theories hitherto mentioned. "At night the respiratory function is modified, the quantity of air exchanged is diminished. This diminution is partly, though not entirely, dependent on a change of the respiratory function of the vocal cords, which, in nocturnal breathing, approach each other more closely than in the waking state, that muscular action by which they are kept apart being relaxed. The more profound the slumber, the greater the relaxation, and the narrower the chink of the glottis. Excess of this relaxation accounts for an asthmatic attack; as naturally the larynx opposes a greater obstacle to the egress than to the ingress of air, the chest soon fills with each inspiratory act. Asthma is thus the result

[1] *Op. cit.*, vol. ii, 460.

[2] Asthma.—*Med. Times and Gazette*, 1863, vol. i, p. 521.

of a disorder of the respiratory function of the glottis. At the same time the fibres of the lungs are relaxed."

Jaccoud[1] was unable to explain an asthmatic paroxysm by either a bronchial spasm or a tetanus of the diaphragm. Both muscular apparatus, he maintained, were equally concerned in its production. Although the innervation of the lungs was still an open question, yet the sonorous and sibilant ronchi seemed sufficiently conclusive of a bronchial stricture.

The contractility of the bronchi, and its dependence upon the vagus, were again inquired into by Paul Bert.[2] This time the sources of fallacy, to which Rügenberg had alluded, were carefully avoided—*i.e.*, the lungs were experimented upon after their removal from the body, and their separation from the adjoining structures. Although at first the results seemed doubtful, in the end the organ was seen to contract, as stated by previous observers, upon electrical irritation, applied either directly to it or to the pneumogastric nerve. But the contractions were so feeble and slow as to induce Bert to think that they had no active share in the mechanism of respiration. Moreover, the paralysis of the bronchial muscles, following the section of the vagus, did not appear to influence in the least the function or nutrition of the lungs.[3] Thus the

[1] *Traité de Pathologie interne.* Paris, 1869, tome i, page 810 *seq.*

[2] *Op. cit.*, p. 371 *seq.*

[3] *Ibid.*, p. 377.

integrity of those muscles, not being indispensable to an efficient ventilation of the lungs, it seemed not unreasonable to conclude that their spasmodic contractions could hardly have the effect generally assigned to them. Bert, therefore, thought it idle as yet to construct upon his experiments a theory of the pathology of asthma.[1]

Biermer, however, relying upon these experiments more than their author did, expressed himself decidedly in favour of bronchial spasm as the cause of asthma.[2] The objections to this theory, upon the ground that the physical signs during an attack seemed incompatible with it, he held to be irrelevant; for the contraction of the thorax and the deficient resonance on percussion, as postulated by Bamberger and Lehmann,[3] could take place only if asthma were a spasm of the alveoli. But Biermer contends against the views of Romberg, Bergson,[4] and Oppolzer,[5] that the spasm was limited to the bronchi[6] and all the symptoms during the paroxysms were readily explicable from the obstacle which it opposes to expiration. According to the law established by Breuer,[7] all respiratory movements are

[1] *Op. cit.*, p. 381.

[2] *Loc. cit.*, p. 1.

[3] Compare pp. 36 and 37.

[4] Compare p. 28.

[5] *Vorlesungen über Spez. Pathol. u. Therap.* Bd. i. Erlangen, 1870, p. 482.

[6] Biermer, *loc. cit.*, p. 7.

[7] *Die Selbsteuerung d. Athmung durch d. n. vagus.* Sitzungb. d. K. K. Akad. d. Wissensch. Bd. 58, Abth. 2, 1868, p. 909 *seq.*

so regulated through the vagus as to conduce to the one salutary end, namely, the efficient ventilation of the lungs. If, as in the present instance, expiration is mainly impeded, this act is prolonged and performed also by the aid of all its auxiliary muscles, whereas the inspiration, which ought to follow in due course, is postponed. By way of reflex, however, the succeeding inspiration is equally increased, merely for the purpose of reinforcing thereby the expiratory pressure. Under these conditions, Biermer adds, the bronchial obstruction partially yields to the forcible inspiratory traction, so as to admit the entrance of air into the lungs, yet the expiratory pressure not only fails to accomplish its useful object, but, notwithstanding the operation of the law which he invokes, it increases the impediment still more by compressing the bronchial tubes instead of the alveoli.[1] Hence the distension of the thorax and all its concomitant symptoms. A tetanus of the diaphragm, however, he remarks, could not persist for several hours without inevitably causing asphyxia, although a similar process may yet take place for days uninterruptedly in the bronchial muscles.

Lebert adverted to the many inconsistencies of that theory. More particularly, he urged that the presence of emphysema during an attack would be quite inconceivable from the account Biermer had given of its origin;[2] nor was it possible that the spasm would be

[1] *Loc. cit.*, p. 7. [2] *Op. cit.*, p. 440.

limited to one portion of the muscles, so as to produce the alleged "valvular" obstruction of the bronchi.[1] Lebert admits a bronchial constriction, as the starting-point of asthma, but the result of the stricture, uniform as it is throughout the air-tubes, consists merely in a cylindrical narrowing.[2] Thus the interchange of gases being interfered with, the ensuing accumulation of carbonic acid in the blood causes the tetanus of the diaphragm and the other muscles of respiration. The opinion of Biermer, he thinks, as to the impossibility of such a tetanus, was deprived of all foundation by his own admission, that in the supposed valvular obstruction of the bronchi the diaphragm was prevented from ascending by the distended lungs; it was certainly immaterial whether a mechanical cause or a spasm interfered with the function of that important muscle. Yet, although inclined to admit an asthma of a purely nervous origin, Lebert confesses that, after an increased experience, he has become cautious with regard to this diagnosis, and year by year meets with fewer instances of the kind. The reason he assigns is the difficulty of distinguishing in a given case of paroxysmal dyspnœa, whether this proceed from a disturbance of innervation or an emphysema of the lungs, which is as yet in its latent stage of development.[3]

According to Leyden,[1] asthma is a peculiar form, if not a symptom, of croupous bronchitis.[2] He maintains that, under circumstances as yet unknown, white blood-corpuscles abundantly emigrate into the bronchi and alveoli; but, instead of being expectorated in the shape of casts, they are retained within the air-passages, and here, in contact with the external atmosphere, disintegrate. In the process of that disintegration there is formed a peculiar kind of octohedral crystals, the points of which irritate the pulmonary filaments of the vagus, and thus induce a bronchial spasm. He bases this opinion upon the presence of those crystals in the sputa, with which, as a rule, all asthmatic paroxysms terminate. Chemical analysis has shown them to be a compound of mucine,[3] and their occurrence in the blood and spleen of leukæmia, as well as in the medulla of bone in leukæmia and health, renders it highly probable that they are derived from the disintegration of the white blood-corpuscles. With regard to the irritation, which the crystals are supposed to exert upon the filaments of the vagus, Leyden has less satisfactory evidence. He assumes such irritation because he had seen them *only* in the sputa of asthmatics, shortly after the paroxysms. The reports of their occurrence also in other diseases do not seem to invalidate the

[1] Zur Kenntniss d. Bronchial-Asthma.—Virchow's *Archiv*, vol. liv, 1872, p. 324 *seq.* [2] Compare Andral and Lefevre.

[3] E. Salkowsky.—Virchow's *Archiv*, p. 344 *seq.*

view he adopted. The cases of catarrhal and croupous bronchitis, and the "*catarrhe sec*" in which they had been met with by Friedreich, Förster, Robin, and Charcot, may, Leyden supposes, probably have been "asthma", an accurate diagnosis being as yet impossible, on account of the prevailing uncertainty concerning this disease. To test his hypothesis he introduced powdered glass into the air passages of animals, without, however, obtaining the expected results. It remains, however, a matter for future consideration whether a process distinguished by a copious exudation of white blood corpuscles is more properly called asthma than croupous bronchitis ; and whether, in the presence of such nutritive disturbances, the bronchial muscles are still capable of a spasmodic contraction. Zenker[1] and Merkel[2] assert that the crystals occur also in the sputa of those cases in which no paroxysmal dyspnœa is observed.

In the theories hitherto mentioned, Professor Weber[3] of Halle, and F. Haring[4] do not find a satisfactory interpretation of all the symptoms of an asthmatic seizure. In their opinion the disease is a vaso-motor neurosis. They attribute, as had been done by Traube,[5]

[1] *Tageblatt d. 45 Versammlung deutscher Naturforsch. u. Arzte in Leipzig*, 1872.

[2] Ziemssen's *Handbuch*, etc., vol. i.

[3] *Tageblatt d. 45 Versamml.*, etc.

[4] *Ueber Bronchialasthma.*—Inaug. Dissert. Halle, 1873.

[5] *Vide* page 32.

the dyspnœa, the râles, and the convulsive respiratory movements to a "catarrhus acutissimus", produced by an unknown derangement of innervation. The obstruction of the nasal passages by the swelling of their mucous membrane is adduced as the proof that a similar process is capable of occluding also the narrow bronchi. They maintain, however, that the hyperæmia is not limited to the ultimate ramifications of the bronchial tree, but extends throughout its entire length, and commences even from within the nasal cavities. K. Störk[1] examined, by means of the laryngoscope, a patient during the paroxysm, and observed the congestion of the trachea.

[1] *Mittheilungen üb. Asthm. Bronchial. u. die mechanische Lungenbehandlung, etc.* Stuttgart, 1875.

CHAPTER III.

DISCUSSION OF THE PREVALENT THEORY.

THE various theories that have been enumerated in the preceding chapter may fitly be divided into two groups—the one based upon the assumption, the other upon the denial, of the nervous origin of the disease. Those comprised in the latter group require no special notice at this place, since from the pathology, about to be given, it will appear how far they are admissible. The present inquiry, therefore, will be limited to the leading principle of the former, and especially to that theory which always has been, and still is, the more generally accepted. Thus the questions proposed for discussion are, whether, as alleged;

A. *Asthma is a nervous affection, and in particular*
B, *a bronchial spasm.*

A. *Is asthma a nervous affection?* Without entering upon the details as to the manner in which this theory has developed, it suffices for the present purpose merely to sum up the arguments adduced in its support.[1] There are cases in which an intense and agonising dyspnœa suddenly arises in the midst of perfect ap-

[1] Hyde Salter, *op. cit.*, p. 31 *et seq.*

parent health, and as suddenly relapses again into a state of ease and tranquillity. The origin of that dyspnœa cannot be accounted for by any of the *recognised affections;* for heart disease, bronchitis, and emphysema, which *alone*[1] are capable of producing such symptom, must be excluded from its causation, on account of the absence of their respective physical signs during, before, and after the paroxysm.[2] Asthma, therefore, appears to be an affection *sui generis*, which, as the absence of physical signs denotes the integrity of the thoracic organs, arises independent of organic lesions. This inference as to the independence of the disease on structural changes tallies with the freedom of respiration, which is immediately restored after the attack. It is confirmed, moreover, by the results of *post mortem* examinations, which are reported to be either completely negative or so varying in character as to preclude the idea of their being the *cause* of a definite disease. Upon the principle that affections which leave no traces of their existence, produce their symptoms through the nervous system, asthma is supposed to be a nervous affection. To the same conclusion tend the causes, symptoms, associated phenomena, periodicity, and treatment of the dyspnœa: all these directly appealing to or acting through the nervous system.[3]

[1] Hyde Salter, *op. cit.*, p. 31.

[2] *Ibid.*, p. 33.

[3] *Ibid.*, p. 25.

It is evident, thus far, that the theory entirely reposes upon the exclusion of organic lesions from the causation of asthma, while the other circumstances just referred to are merely subservient to the main issue. If, therefore, it can be shown that the dyspnœal attacks are *not* independent of structural lesions, the whole fabric must necessarily collapse. Now, on close examination, it cannot fail to be seen that all the facts and inferences, as stated above, are erroneous.

1. *The inference from the absence of physical signs.* In the present state of pathology, it is superfluous to say that there are other *recognised* affections besides heart disease, bronchitis, and emphysema, capable of producing sudden and intense dyspnœa. Although their diagnosis may be surrounded with certain difficulties, yet their existence is therefore not the less real. But, be this as it may, not even bronchitis, heart disease, and emphysema, can be excluded upon the grounds assumed. A patient may daily expectorate several spittoonfuls of mucus, and yet the most practised and most careful observer may fail to detect, by means of percussion and auscultation, the least trace of disease. The reason is that the mucus forms and stagnates in a bronchus or bronchi, situated at some distance from the surface of the chest, in which position the adventitious sounds are, as the bronchial respiration is normally, obscured by the intervening

healthy tissue of the lungs.[1] So also in the case "of plastic exudation thrown around the tube", the contraction may indeed be irremediable, yet the sibilus is by no means unvarying,[2] but for a time the wheezing completely disappears, and only returns with a temporary swelling of the mucous membrane, increasing the stenosis.[3] Again, many cases of emphysema exhibit no typical symptoms, as described in text books. The long and paralytic thorax presents no barrel-shaped distension, but maintains its shape, in spite of extensive emphysema. In the atrophous forms of the disease, such as occur in old age, or as a sign of premature senescence in advance of the involution of all other organs,[4] or even in childhood, in consequence of inflammation,[5] there are not only *depressions* of the thorax, but also *reduction* of all its diameters.[6] Percussion and auscultation are equally insignificant in those cases ; the former is not always hyper-resonant or tympanitic, while the respiratory murmur is often as clearly heard

[1] Wintrich, *op. cit.*, p. 169.—C. Gerhardt, *Lehrbuch d. Auscult. u. Percuss.* 2 Aufl. Tübingen, 1871.—Lebert, *op. cit.*, pp. 54, 96, 273. [2] Hyde Salter, *op. cit.*, p. 37.

[3] Biermer, Krankh. d. Bronch. u. d. Lungenparench.—Virchow's *Handb.*, Bd. v, Abth iv, 1865, p. 776.

[4] Rokitansky, *Lehrb. d. pathol. Anat.*, Bd. iii, 3 Aufl. Wien, 1861, p. 48.

[5] Buhl, *Lungenentz. Tubercul. u. Schwindsucht. Zwölf Briefe.* München, 1872, p. 22.

[6] Niemeyer-Seitz, *Lehrbuch*, etc. 9 Aufl. Berlin, 1876, p. 125.

in this disease as it is in health.[1] The same may be said of affections of the heart. The mere existence of certain abnormal physical signs does not warrant the assumption that textural change exists.[2] And, conversely, the total absence of physical signs does not prove the heart to be in a perfect state of organic soundness.[3] A young woman applies at the hospital, suffering from an attack of intense dyspnœa. No cause of its origin is discoverable on the repeated and most attentive examination of the chest. In a few days the dyspnœa completely subsides, and the patient is discharged as cured. But in the course of several months she returns to the hospital, now with the unmistakable signs of chronic mitral disease. Lebert[4] properly concludes that the previous attack of dyspnœa was produced by an embolus, which had been washed from the right auricle into the pulmonary artery. An observer, biassed in favour of certain theories, might, however, have judged of this case in a very different manner. It is readily conceivable what would have been the diagnosis. Now such and similar instances may be adduced in large numbers; but those already men-

[1] Niemeyer-Seitz, *op. cit.*, p. 126, *seq.*

[2] Walshe, *A Practical Treatise on the Diseases of the Heart*, etc. Fourth edition. London, 1873, p. 151.

[3] *Ibid.*, p. 152.

[4] Krankheiten d. Blut. u. Lymphgef.—Virchow's *Handbuch.* Bd. v, Abth. II, 2 Lief. 2nd edition, p. 488.

tioned will suffice to show that anatomical changes may exist without attendant physical signs. The fact is that auscultation and percussion reveal only "coarse" organic lesions, and these, moreover, under special favourable conditions. But as little as the presence of physical signs alone explains the actual morbid condition, so little does their absence, when circumstances are less suitable for their production or perception, prove the integrity of the organs. This comparative shortcoming, if so it may be called, detracts in no way from the importance of auscultation and percussion. After all, these methods are but one, though an invaluable, aid to diagnosis. They do not, however, supersede, but supplement the other results of clinical observation, and only by their combined consideration is it possible to arrive at true conceptions of the nature of a disease. Hence, the seeming inadequacy of physical signs during and after an asthmatic paroxysm, is insignificant in itself. A more comprehensive view of the case, one not limited to prominent symptoms, but including all its symptoms, its antecedents, and sequelæ, fully accounts for its pathology. Such a survey shows that asthmatics are "generally miserable looking wretches, round-backed, cyanosed, veiny, and thin",[1] or prone to obesity at an early period of life—a sign of premature senescence. Ninety per cent. of them have, previous to their first attacks, suffered from

1 Hyde Salter, *op. cit.*, p. 101.

inflammatory affections of the lungs, from which, although the acute symptoms have subsided, they have never completely recovered; and after but a short persistence of the asthma, there invariably appears emphysema—a disease, so far as its pathology is known, essentially chronic in its development. Here, therefore, is a chain of evidence complete in itself. And, in the face of this evidence, it would be casting aside the accumulated experience of centuries to assert that, with such antecedents and sequelæ, the respiratory organ, constantly the seat of functional disturbance, is, nevertheless, in a sound condition, because auscultation and percussion show nothing to the contrary.

A method of physical exploration, apparently more delicate than auscultation and percussion, serves to confirm this conclusion. By means of manometric measurements, Waldenburg[1] found that in certain affections of the lungs, the proportion existing between the forces of inspiration and expiration is disturbed in a definite manner. Whilst in health the force of expiration exceeds that of inspiration, in disease the one or the other may be *relatively* deficient. Thus emphysema is distinguished by a diminution of the expiratory pressure, whereas inspiration is either normal or even excessive. Waldenburg observed this

[1] Die Manometrie d. Lungen, oder Pneumatometrie, etc.—*Berlin. Klin. Wochensch.*, No. 45, 1871.

expiratory deficiency also, although in a lesser degree, in those recently subject to asthma, but who in the intervals between the attacks enjoy perfect health, and in whose organs of respiration and circulation no trace of disease is discoverable by means of percussion and auscultation. The asthmatic paroxysms cannot so rapidly injure the elastic tissue of the lungs, and the inference, therefore, is not unfounded that this pathological condition is antecedent to the attacks. Whatever imperfections there may be in the method itself, they do not invalidate the results obtained thereby, these being relative and not absolute.

2. *Freedom of respiration between the attacks.* Complete freedom of respiration is not inconsistent with such impairment of the elastic tissue of the lungs, for, even in many cases of fully developed emphysema, there is no dyspnœa, so long as there is no bronchitis. The statements, therefore, which asthmatics delight in making, that, in the intervals between the attacks, their respiration is free and easy, and they themselves are capable of undergoing great physical exertion, are fully deserving of credit. Yet any inference from them as to the integrity of their respiratory organs would be erroneous. Many consumptives in the same way declare that they never cough or expectorate, and, when caught in the act, maintain that this is not worth their attention. Their lungs, they say, are perfectly sound, only their digestion is de-

ranged. The fact is, one entire lung may be destroyed by disease, and yet the patient may be able to enjoy hunting and shooting.[1] Again, those suffering from affections of the heart and large vessels, that may prove fatal at any moment, frequently present all the appearance of health, and are able to attend to their usual occupations without any inconvenience. So convinced are they of the soundness of their organs, that they express their petulant annoyance when the chest is subjected to a physical exploration.[2] It is fortunate for persons thus affected to be ignorant of the dangers that surround them. But, as sensations do not correspond to the actual condition of the organs, the unembarrassed respiration of the asthmatic does not prove that his lungs are healthy. Indeed, his very looks, while professing his ability to breathe as freely as anybody, belie his words. Every few seconds he is forced to interrupt that profession by a deep inspiration; and the peculiar attitude he assumes when sitting down, and the prominence of the sterno-mastoid muscles, sufficiently show his constant respiratory efforts. Sir Thomas Watson[3] attributes the present feeling of ease and comfort to the remembrance of past suffering. But there is also a physio-

[1] Walshe, *A Practical Treatise on Diseases of the Lungs.* Fourth edition, p. 536. London, 1871.

[2] Idem, *Diseases of the Heart*, &c., *loc. cit.*

[3] *Lectures on the Principles and Practice of Physic.* London, 1871, p. 394.

logical explanation of the phenomenon. In health and quiet respiration only one-third of the lungs is employed for the performance of the function.[1] Subjective dyspnœa takes place only when the respiratory surface is reduced below that portion. Moreover, the organism possesses a certain power of accommodation to a diminished supply of oxygen, provided that diminution takes place gradually. The chemical changes then become less active, nutrition is perverted, and the sensitiveness of the centre of respiration is so greatly impaired as not to be affected by an accumulation of carbonic acid in the blood, which, in the healthy individual, would rouse it to a violent activity.[2] Thus all this shows that health is but a relative condition, and that a state unbearable to the one may be, in the other, unaccompanied by any inconvenience.

3. *Post mortem examinations;* A. *Negative results.* With few exceptions the alleged negative results of *post mortem* examinations date from a time when pathological anatomy was as yet in its infancy, and the term asthma was used still more promiscuously than it is now. Thus, as already stated, Andral and Laennec had, in all probability failed to discover the embolon in the pulmonary artery, and therefore erroneously

[1] Cruveilhier and Broussais, quoted by Paul Niemeyer, *Handb. d. Percuss. u. Auscult.* Bd. ii, Abth. ii, p. 72. Erlangen, 1871.

[2] Claude Bernard, *Leçons sur l'action des substances toxiques et médicamenteuses.* Paris, 1857.

inferred the absence of organic lesions. Other negative results may possibly be explained in a similar manner, for there are morbid conditions capable of producing intense, and even fatal dyspnœa, yet so fleeting in their nature as to leave no traces of their destructive operation. Œdema of the glottis and croup of the bronchi are, among others, instances of the kind. In the former, the vessels contract after death, and expel their contents ; in the latter, the softened pseudo-membranes are either expectorated or appear merely as the ordinary mucous secretion. If the clinical observation was at all imperfect, or under the bias of a certain doctrine, it is easy to conceive of what nature were the cases in which asthma was not accounted for by the examination after death. But there is positive evidence that many of those autopsies were not sufficiently exhaustive. They were performed, not with the intention of obtaining a clue to the cause of the dyspnœa and death, but with the desire of proving a preconceived theory. One example will suffice to show the justice of this remark.

"A lady had been, during the last three years of her life, tormented by periodical attacks of dyspnœa. She was seen, in consultation with her ordinary medical attendant, two days before her death. She was then sitting in bed, presenting all the symptoms of dropsy. She vomited all she took, and was therefore in a state

of great prostration. Her condition was already beyond the resources of medicine."

"On *post mortem* examination the lungs were found to be adherent at some places, but otherwise healthy. The intestines, and especially the jejunum, were swollen and greatly distended with gas, but contained no fæces. The spleen and liver were healthy, the enlarged gall bladder enclosed a calculus about two drachms in weight."[1]

Apparently none of the organs, the diseases of which alone could have explained the dropsy, the vomiting, the dyspnœa, and death, were examined. Théry was satisfied with the anatomical diagnosis of *gaseous distension of the intestines*,[2] and at once concluded that the case was one of nervous asthma. He, moreover, collected sixty-five similar cases, upon which he based his opinion that organic lesions were not concerned in the causation of asthma. This fact has, doubtless, escaped the attention of Biermer, else it is impossible that he would have referred to Théry as "one who well knew the clinical features of essential asthma, and who, from the study of them, learnt that they can be explained only by the assumption of a spasmodic element."[3]

Thus the reports of a former date are not sufficiently reliable to warrant any conclusion. At present, when

greater care is bestowed upon pathological anatomy, and the limits of asthma are more restricted, the fatal issue of the disease is so rare, as to induce Hyde Salter altogether to deny its occurrence.[1] The fact is that, for some unaccountable reasons, asthma is at present observed chiefly in private practice, where *post mortem* examinations are seldom or never performed; while cases admitted into hospitals with that diagnosis generally cease to be regarded as such during observation; at all events after death. Thus it happened in the following case, observed by Professor Rienecker.

"Michael Mayer, aged 76, tall and well built, a carpenter by trade, applied at the hospital in February 1850, suffering from a very severe bronchitis. His story was that, with the exception of repeated attacks of dysuria and gastric catarrh, he had been quite well until he caught the present 'cold'. The bronchitis was soon complicated with violent asthmatic seizures, which, however, presented the usual symptoms. During the attacks the pulse was intermittent, and the temperature greatly diminished. The catarrhal symptoms gradually abated, and with them also the dyspnœal paroxysms. The patient so far recovered as to be able again to follow his ordinary employment, but a slight dyspnœa remained. In the middle of May he had another attack of asthma, again accompanied by catarrhal symptoms. As usual there was the feeling

1 *Op. cit.*, p. 154.

of constriction of the chest, great anxiety, coldness of extremities, great muscular debility, and irregularity of the pulse. The lungs gave everywhere a full and sonorous percussion note, and there were a few mucous râles at the bases. The cardiac impulse was almost imperceptible, the area of the heart not enlarged, and the sounds were not altered. The exhibition of stimulants and expectorants produced some amelioration. The attacks, however, returned at intervals. On the 6th of June the patient awoke with another attack, which, after a protracted agony of about twelve hours, terminated fatally the next day."

The clinical diagnosis was *bronchitis with asthma*. But the *post mortem* examination revealed the cause of the dyspnœa and the death.

"The second divisions of the pulmonary artery contained plugs of different ages. Both the recent and the older emboli were prolonged into the arterial branches. Those of older date were dry, discoloured, and in some places adherent to the vascular walls, and softened in the centre; the more recent ones were smooth, dark red, and not adherent. Corresponding thrombi, contrasting with each other in the manner just described, were found also in the right auricle. The right side of the heart was dilated and hypertrophied. There were, moreover, chronic bronchitis, with dilated bronchi, gastric catarrh, fatty degeneration of the liver, an old empty hernial

sac in the median line, and two inguinal herniæ."[1]

Modern literature contains, so far as I am able to ascertain, only two cases in which the asthma was *not* accounted for by the *post mortem* appearances. The one is reported by Phœbus.[2]

"A solicitor, aged 59, had, when a child, been suffering from scrofulosis, and subsequently from frequent attacks of bronchitis. At the age of twenty-five he became subject to an habitual headache; at twenty-six he had hay asthma for the first time, the chest symptoms being particularly marked. At noon on the 31st of May 1860, while still subject to cough, he took a warm sulphur bath, which he left, however, in less than a quarter of an hour. The attendant at the baths then found him with distorted face, very anxious respiration, and severe cough. The patient could speak only with difficulty, intimating that he wished for a doctor. But he died in ten minutes, before aid arrived. Shortly after death a quantity of frothy fluid oozed from his mouth.

"On examination, eighteen hours after death, the lungs were found to be distended, of dark colour, and œdematous at some places. The trachea and bronchi

[1] Virchow, *Gesammelte Abhandlung. z. wissensch. Mediz.* Frankfürt, 1856, p. 356.

[2] *Der typische Frühsommer-Katarrh od. d. sogen. Heu-Asthma.* Giessen, 1862, p. 69 *seq.*

were filled with a frothy fluid, the bronchial mucous membrane was tumid and reddened in spots. The pleural cavities contained a quantity of serum. The heart was covered with fat, but distinctly hypertrophied. The cerebral vessels were over-filled, and the cerebral cavities contained fluid blood."

As the state of the kidneys is not mentioned in the report, it is not unreasonable to infer that they had not been examined. But whether or not the hypertrophy of the heart, the bronchitis and cerebral hæmorrhage were all connected with renal disease, the case by no means proves that the habitual cough and dyspnœa, from which the patient had suffered for thirty years, was not due to organic lesions.

The other case[1] above alluded to, was that of a healthy labourer aged 36, who was admitted into the hospital suffering from intense dyspnœa. Seven weeks previously he had been suddenly seized with cough and dyspnœa ; but, while the former gradually subsided, the latter so greatly increased as to force him to keep in bed. It was noticed that the asthmatic paroxysms, which continued during his stay at the hospital, were ushered in by a kind of epileptic cry ; and, at the height of the dyspnœa there was loss of consciousness, distortion of the face, opisthotonos, and tetanus of the muscles of the limbs. On recovery the patient anxiously preserved the horizon-

[1] Bamberger, *loc. cit.*, p. 102.

tal posture in bed, for fear lest the attempt to move should cause a recurrence of the same symptoms. Apparently, this fear was not unfounded, for the examination or even the observation of the patient seemed to provoke a fresh attack. Three days after admission, the patient died; and the autopsy revealed nothing abnormal, except congestion of the lungs and hypertrophous dilatation of the right ventricle. This case, however, does not bear upon the matter at issue; for, though the intense dyspnœa was produced, as Bamberger states, by tetanus of the diaphragm, yet the tetanus was general, and all the other symptoms widely differed from those that are generally considered as distinctive of asthma. So long as the diagnosis of this affection is wholly based upon the combination of such phenomena as the dyspnœal seizures and the intervals of free respiration, it is inadmissible to adduce and argue upon cases that do not present the same clinical features. Disregard of these characteristics would render asthma synonymous with dyspnœa, and would serve to still more complicate the question of its nature.

B. *Positive results.* The fact thus remains that, in the vast majority of instances, pathological conditions are, and have been, met with on *post mortem* examinations. Their alleged variety, however, applies only to cases of a former date, and proves not that asthma is independent of anatomical lesions, but that the term

had been indiscriminately employed. At present when, as already mentioned, the limits of the disease are greatly restricted, the organic complications are consequently also of a more definite character, and more constant in their appearance. Even the pure and idiopathic asthma—if it is at all severe—is soon accompanied by structural changes which, according to the prevalent doctrine, are believed to be the *consequences* and not the *causes* of the dyspnœal seizures.

Yet, the principle upon which this opinion is based is quite inconsistent with the teaching of modern pathology. No shadow of a proof can be adduced to show that, in any case, derangement of function causes derangement of texture. If the functional disturbance apparently precede the organic lesion, the latter is merely at the time inaccessible to diagnosis. The known pathology of the presumed consequences of asthma is, moreover, opposed to that view. Thus, it is said that the earliest complication of the dyspnœal attacks is thickening of the bronchial tubes, due to hypertrophy of their muscles —a process that arises from their exalted activity during the spasm, according to the law that determines the hypertrophy of the blacksmith's arm, or that of the bladder and the heart in urethral stricture and valvular lesions respectively.[1] That there is often in asthma a thickening of the bronchial walls is unquestionable ; but that the thickening

[1] Hyde Salter, *op. cit.*, p. 156.

consists of an increased development of the circular fibres has been asserted upon inferential grounds, without actual inspection of the organ.[1] Still, the possibility of such occurrence cannot be denied. For, though the great majority of histologists attribute that thickening to the formation of connective tissue, that even destroys the contractile elements, yet a muscular hypertrophy, if not of the bronchi, at any rate of the pulmonary tissue has been observed in isolated cases of brown induration of the lungs,[2] and in that form of parenchymatous pneumonia described by Buhl as "muscular cirrhosis".[3] But, if these processes be associated with asthmatic paroxysms, the latter are surely not the cause of the former. Besides, the law that increased activity of a muscle causes an increase of its nutrition is inapplicable to asthma. If the muscles of the blacksmith's arm enlarge, it is because a *healthy* function is daily continued in a *healthy* individual. But a bronchial spasm, occurring at more or less distant intervals, would be a *morbidly altered* function, which is not known to have that effect. Certain it is that an epileptic may be subject to fits every day of his life, and yet no one of his muscles on that account undergoes a hypertrophous development. The case of the "irritable bladder" affords but a doubtful

[1] Hyde Salter, p. 371.

[2] Rindfleisch, *Centralblatt f. d. Mediz. Wissensch.*, 1872, No. 5, p. 65.

[3] *Op. cit.*, p. 58.

proof, inasmuch as the "irritability" may be the index as well as the precursor of the hypertrophy. The same applies to the idiopathic hypertrophy of the heart from nervous palpitation. Although the excessive action *seems* to be the immediate cause of the cardiac enlargement, yet, the structural change, as well as the nervous palpitation, are probably always the consequences of a congenital narrowness of the arterial system.[1] Nor has the theory of final causes — that the muscles may be equal to the work[2]—any bearing upon the present question. If the bladder in urethral stricture, and the heart in valvular lesions, increase in size and force, such increase is a sign of a vigorous constitution; it is a *salutary* compensation, intended to counteract obstacles to the functions, the free performance of which is necessary for the maintenance of life. But the hypertrophy of the bronchial muscles would only augment the evil. "One certain result of this hypertrophy is a permanent thickening of the bronchial walls and consequent narrowing of their calibre; and one possible result is a greater disposition on the part of the hypertrophied muscle to take on a state of contraction. To the former, perhaps, is in part due that slight constant dyspnœa which is so disposed to de-

[1] Virchow, *Ueber d. Chlorose u. die damit zusammenhängenden Anomalien im Gefässapparate etc.* Berlin, 1872.

velope itself in asthma; to the latter, the increasing tendency to and frequency of spasm which characterises some cases."[1]

Again, the curvature of the spine, the so-called asthmatic *physique*, is attributed to the forcible contractions of the *erector* muscles of the back, which, while engrossed with their violent respiratory labour, permit the vertebral column, thus unsupported, to fall forward. The vertical pressure which the bodies of the vertebræ exert upon each other is said to cause absorption of their anterior portions, and consequently the deformity.[2] From this deformity alone, Hyde Salter relates, he often diagnosed spasmodic asthma by merely looking at persons whom he passed in the streets. Still, as there are other diseases capable of producing precisely the same symptoms, and as, moreover, he had avowedly never examined a spine *post mortem*,[3] his diagnosis as well as his pathology are not supported by satisfactory evidence. Indeed, it is difficult to conceive that the increased activity of the muscles, whose special function is to *extend*, should yet permit the *flexion* of, the spine. But, be this as it may, the stooping that really occurs during the paroxysms is hardly sufficient to account for the spinal curvature. The case which Hyde Salter refers to,[4] and Dr. Theodore Williams repeats, by no means proves

[1] Hyde Salter, *op. cit.*, p. 156.
[2] *Ibid.*, p. 174.
[3] *Ibid.*, p. 175.
[4] *Ibid.*, p. 175.

the point.[1] "A man in the prime of life, with an erect posture, who had never had anything the matter with his spine, was seized with severe *rheumatism* in the muscles of the back; and the pain was so aggravated and rendered so intense by attempting to stand upright, that the muscles refused to perform their function; the unsupported spine bent forwards as far as its elasticity would permit, the back became round, and the gait stooping like that of an old man. This went on for some months, without any decided curvature appearing; at length, distinct and gradually increasing curvature manifested itself, involving the seventh and eighth dorsal vertebræ to such an extent as to show that their bodies must have undergone some change of shape. The patient recovered from his *rheumatism*, regained the power of supporting his back, the curvature was arrested, but it has never disappeared." Now, so long as there is nothing but pain in the back, rheumatism of the muscles is, indeed, a very probable diagnosis; yet the subsequent appearance of a curvature points to disease of the bones. The fact is, faulty attitudes lead to spinal deformities only when the pressure is more or less permanent, and when the ossification of the vertebral column is still incomplete. Asthma, however, if at all operative, furnishes merely the mechanical cause; and, since the curvature ap-

[1] Clinical Lectures on Spasmodic Asthma.—*Lancet*, Sept. 6, 1873, p. 325.

pears with equal frequency in the severer as well as in the milder cases, in which the attacks are of short duration and separated by long intervals, it therefore follows that the bones must be of an abnormal softness. This, indeed, seems to be the true solution of the question. For not only the spinal deformity, but also the shape of the head and that of the limbs show that the asthmatic is *rickety;* so that his dyspnœa and his *physique* are co-ordinate results of the same constitutional cause.

And, lastly, the pathology of emphysema is equally inconsistent with the prevailing opinion. Asthma can produce a loss of elasticity and a permanent distension of the lungs only by the disturbance of the respiratory pressure during the paroxysms; yet such mechanical force is ineffective unless there be a predisposition inherent in the lungs. The facts, that at times emphysema spontaneously arises, as it were, and precedes the bronchitis, the cough, and the dyspnœa; at others rapidly and extensively developes when the disturbance of the respiratory pressure has been but slight and of short duration; and again at others gradually appears and to a limited extent when the same causes had been persistent and powerful; all these forcibly show that certain nutritive changes of the pulmonary tissue are concerned in the production of that disease.[1]

[1] Biermer, Virchow's *Handb.*, Bd. v, p. 789 *seq.*; Niemeyer-Seitz, *op. cit.*, p. 120 *seq.*; Lebert, *Klinik der Brustkrankheiten*, Bd. i. p. 399 *seq.*

The nature of these changes is as yet unknown; and for the present purpose it is immaterial whether it consist in an extension of the catarrhal inflammation from the bronchi to, or in a primary disease of, the alveolar walls,[1] or in thrombosis of the pulmonary vessels, followed by softening and absorption of the elastic tissue.[2] It suffices for the present purpose to state the fact that even if asthma is pure and idiopathic, it is soon complicated with emphysema; and as the dyspnœal seizures furnish only the mechanical element, it therefore follows that under these conditions the lungs cannot be healthy, but must be the seat of pathological changes.

Hence the principle that "diseases which leave no traces of their existence produce their symptoms through the nervous system,[3] is, whatever its merits may be, inapplicable to asthma; for though in a few and rare instances the lungs *may seem* perfectly sound, still there is no evidence that they *are so*, whereas the symptoms and the results of *post mortem* examinations tend to opposite conclusions.

It is futile to settle the nature of a disease without the concurrence of pathological anatomy. The experience of the former systems of medicine abundantly

shows that such attempts have invariably ended in failure. Although the progress of medicine may be supposed to afford at present a greater prospect of success than was then attainable, it is but necessary to remember, in order to dismiss this view, that the progress itself is mainly due to the cultivation of that branch of science which would here be excluded. All the other data from which the nature of a disease might be inferred, are not as yet sufficiently reliable for the purpose; and it is surprising, therefore, to find that, nevertheless, in the case of asthma, so much undue importance has been attached to them.

4. *Causes.*—Amongst the alleged causes of asthma —the list of which includes almost every thing and circumstance in life—there is admittedly not one that can be declared as inevitably provocative of the dyspnœa; on the contrary, so uncertain is their operation, that what will bring on an attack in one case will be harmless in another; what will be fatal in the other will be innocuous in the one;[1] nay, what will *produce* it in the one will *cure* it in the other.[2] Such inconsistencies, incompatible as they are with the very conception of cause, would, even in the lax logic of pathology, plainly indicate that no relation existed between two events. But here the *capriciousness* of asthma is said fully to account for them, and to prove, moreover,

in the clearest manner, the nervous origin of the disease.[1]

Yet, if etiology in general is acknowledged to be the most obscure department of medical science,[2] that of asthma, in particular, certainly makes no exception to this rule. The fact is, there is hardly one instance in which the real cause of the dyspnœal seizure has been ascertained, whereas, doubtless, in the vast majority of cases, circumstances that accidentally *preceded* were supposed to have *produced* the attack. To a great extent, the obscurity on the subject and the confusion resulting therefrom are mainly attributable to the subtilty of the exciting agents; for since their presence can, as a rule, be detected only by long and careful observation or merely by accident, it has, for obvious reasons, generally escaped notice. But not the least of the prevailing uncertainty on the matter is due to the readiness with which credit has been given to whatever the patient or his friends imagined as the *causes* of the attacks. Naturally, the patient whom the agonising dyspnœa rouses from his sleep, and his friends, the sympathising witnesses of his sufferings, are above all anxious to discover the source of such formidable accidents, in order to avoid their repetition. Actuated by the desire to gain this knowledge, they are unmindful of the fallacy that attends the *post hoc ergo propter*

[1] Hyde Salter, *op. cit.*, p. 107.

[2] Wagner, *Handbuch. d. allg. Pathologie.* Leipzig, 1868, p. 57.

hoc, and admit, without discrimination, as a cause any thing or circumstance, real or fancied, that satisfies their own minds on the subject. The information thus obtained as to the origin of the dyspnœal seizures need not be altogether useless. But the opinions of the informants must be strictly taken for what they are worth. Without a careful scrutiny, they are not available for scientific purposes; yet this scrutiny has generally been neglected, and the result is that upon mistaken facts have been founded still more mistaken theories. The following instances will serve to confirm the view here adopted.

A patient, Laennec relates, had, while about to sleep in a dark room, an attack of asthma, but immediately obtained relief on relighting the night lamp and opening the door so as to admit the access of fresh air. Darkness is thus believed to be a cause of dyspnœa, and as such a cause can act only through the nervous system, asthma is, therefore, a nervous affection. Hence Lebert advises his patients to have as many candles or lamps as possible in their bedrooms in order to avert or abbreviate the asthmatic paroxysms.[1] But throughout the range of physiology, there is not one ascertained fact that could be adduced in support of that theory. Light, indeed, is known to exert a certain influence upon the excretion of car-

bonic acid, and the experiments of Moleschott[1] show that frogs, kept in the dark or blinded, exhale less of that gas than they do when exposed to the light or with unimpaired vision. Yet no straining of these experiments can account for the occurrence of dyspnœa in man under the conditions above mentioned. But there is really no valid reason for assuming a causal connection between the darkness and the asthma. Other circumstances may have equally, if not more, contributed to that effect, and though at present it is possible only to surmise the origin of the attack, still it seems not improbable that the dyspnœa arose from the inhalation of acroleine, carbonic oxide, and carbonic acid,—products derived from the combustion of the glycerides which escape into the air after the imperfect extinction of candles and lamps. At all events, this much is certain, that these substances, and especially the former of them, are highly irritative of the conjunctiva and respiratory mucous membrane,[2] and are reported even as having caused asphyxia.[3]

Again, subtle atmospheric conditions are generally regarded as the most frequent causes of asthma; and, as their hurtful influence is attributed to imperceptible variations of temperature, pressure, or moisture, it is

[1] *Wiener Mediz. Wochenschrift*, 1855, No. 43, p. 682.

[2] Strecker, *Organ. Chemie.* Braunschweig, 1867, p. 176.

[3] Hermann Eulenberg, *Die Lehre von d. schädlichen und giftigen Gasen.* Braunschweig, 1865, pp. 154 and 240.

almost necessary, in order to account for their action, to assume an increased irritability of the nervous system, which registers, as it were, such slight changes of the barometer. But the question assumes a very different aspect if the inorganic and organic impurities of the atmosphere are allotted their due share in the production of the dyspnœa. Then, the cases constantly alluded to,—as that of one patient being seized with asthma in a particular room, and that of another suffering in the pure air of an open space, but being free from the disease in the filthy neighbourhood of crowded cities—are deprived of the mystery that seemingly surrounds them. It is well known that the air contains in variable quantities, at different places and at different times, not only gaseous irritants, but also fragments of stone, of glass, and of iron, fibres of wool and linen, and numerous fungi and pollen grains. It is equally well known that all these substances enter the air-passages, especially if the patients breathe through the mouth instead of the nose ; and that those foreign bodies are capable of producing, under favourable conditions, hyperæmia and inflammation of the bronchial mucous membrane. The inhalation of a small quantity of Penicillium glaucum is reported as having given rise to an intense bronchitis.[1] The sneezing fits from which Trousseau

[1] Ch. H. Blackley, *Experimental Researches on the Causes and Nature of Catarrhus Æstivus.* London, 1873, p. 58.

suffered in rooms in which violets were kept[1] were probably due to the inhalation of Puccinia. For, so far as it is known, this fungus grows by preference upon those flowers,[2] and its presence in the thick green mucus discharged from the nose has been observed by Virchow.[3] That among the number of the individuals who expose themselves to the same irritants, only some are affected, whereas others escape unhurt, must be attributed to the peculiar predisposition of the former. According to experimental inquiry, injection of fungi into the air-passages produces no effect upon the bronchial surface[4] unless there be a previous inflammation—a condition rarely wanting in the asthmatic. With these data, the occurrence of the dyspnœa in the cases above-mentioned admits of a ready solution. A house may be surrounded with the purest atmosphere, yet in it there may be one single room in which the air swarms with organisms or organic irritants. Pasteur made a similar observation in the inn on the glacier. The asthmatic seizures in the open space and their subsidence in crowded neighbourhoods are, no doubt, due to the presence and absence of pollen grains in the air of the respective places. But

[1] *Op. cit.*, tom. ii, p. 449.

[2] Rudolf Maier, *Lehrb. d. allg. patholog. Anatom.* Leipzig, 1871, p. 574.

[3] *Archiv*, vol. ix, 1856, p. 578.

[4] Joh. Popoff, *Zur Frage über Pneumonomycosis;* Stricker's *Medic. Jahrb.* 1872, Heft iv.

the presence or absence of these and similar irritants can be ascertained only by microscopic examinations; and when this means of research is neglected the observation is defective, and the conclusion based thereon erroneous. Thus it is with the report of Dr. Theodore Williams, that "some passengers in the train with him were seized in certain regions through which they passed with sharp attacks of asthma, and then, as they left these places, the breathing of these people became natural".[1] Admitting the fact, the implied inference that the dyspnœa was due to subtle atmospheric conditions and consequently to a greater irritability of the nervous system, is wholly without foundation. In reality, some rails wear down more readily than others, so that plates of iron are torn up and rubbed off by the pressure of the train.[2] Particles of that kind float in the atmosphere of railway carriages,[3] and lacerate, when inhaled, the surfaces of the bronchi.

Often in the acts of swallowing, talking, or laughing, foreign bodies of various sizes enter the air-passages, and such accidents are most likely to occur in the emphysematous, in whom the glottis is abnormally capacious or its closure imperfect in consequence of chronic catarrh. But, as the entrance is effected un-

[1] *Proceedings of the Royal Medical Society of London*, vol. i, p. 95.

[2] Robert Angus Smith, *Air and Rain*. London, 1872, p. 449.

[3] Joseph Sidebotham, *Schmidt's Jahrb.*, 1874, p. 114.

perceived by the patient, the nature of the dyspnœal seizures, produced by the temporary obstruction of a bronchus, remains unknown, unless casually revealed. In illustration of this fact, it is but necessary to refer to the case of Dr. Nooth,[1] who had suffered for years from "nervous asthma", until he expectorated a shot, which doubtless had acted as a ball-valve within the air-tubes. So also a physician was subject to nocturnal attacks of dyspnœa so long as he slept upon a certain pillow, but obtained relief on exchanging this for another.[2] The cause of these asthmatic seizures was not explained, and their occurrence, therefore, still forms the fruitful theme of speculation. But in a very similar instance the mystery was solved in a satisfactory manner. A healthy brewer was one night roused from his sleep by a violent fit of coughing. For six months, the nocturnal attacks returned without known or appreciable causes. At last, the dyspnœa ceased upon a copious expectoration, in which a small feather was found embedded in thick mucus. The patient then completely recovered.[3] Now, if the sputa had not been examined, this case also would have led to very erroneous conclusions. But,

[1] *Transactions of the Society for the Improvement of Medical Knowledge*, vol. iii.

[2] Théry and Ramadge, quoted by Sée, *loc. cit.*, p. 648.

[3] Anton. Biermer, *Die Lehre vom Auswurf.* Würzburg, 1855, p. 64.

in the existing treatises on asthma, no mention is made that, even in those cases in which such a cause of the dyspnœa may be suspected, the necessary examination had been instituted. On the contrary, "nuts, almonds, and cheese", particles of which appear in the sputa for days after their consumption, are considered to be "highly asthmatic" articles, which contaminate the sanguis cibi, thereby irritating the pulmonary filaments of the vagus.[1] And, again, because a lady, possessed of a good appetite and good digestion, "is seized within *a few minutes* after the commencement of her meals with the tight constrictive breathing peculiar to asthma", Hyde Salter concludes that "this case shows a morbid sensitiveness which exalts that into a stimulant which should not be a stimulant, so that the nervous system registers, as it were, on the bronchial tubes, *changes in the constitution of the pulmonary blood*, of which it should be unconscious".[2] But, whether in a few minutes the constitution of the pulmonary blood could be so altered, is as yet even more than doubtful. If it were permissible to conjecture, from the observation of voracious patients, the cause of the dyspnœa in the present case, the aspiration of pepper or similar substances would readily account for the symptoms described.

And, lastly, with regard to the alleged emotional asthma, it must be admitted that mental excitement

[1] Hyde Salter, *op. cit.*, p. 46. [2] *Ibid.*, p. 47.

may possibly, in persons with erethism of the heart, induce hyperæmia of the lungs, and consequently dyspnœa; but in the great majority of instances in which psychical stimuli were supposed to be operative, the real causes of the asthmatic seizures escaped notice. If the hysterical young lady, who had been subject to hay asthma for seven years, was seized with her complaint on inspecting a highly finished picture of a hayfield, the mere impression could not, from what is now known of the nature of the affection, have been, as supposed, the exciting cause.[1] Nor is it credible that because a French officer was deeply moved at the sight of the enemy before the gates of Paris, the emotion should have been powerful enough to give rise to the recurrent attacks of asthma of which he was not relieved even by a long sojourn in the south of France.[2] In this, as well as the preceding case, circumstances besides those mentioned must have, though unsuspected, intervened in the causation of the disease. How easily such circumstances may be overlooked, and what mistakes such oversight leads to, is evident from the following instance. A medical man lost his wife, and sought relief of his sorrow by spending daily several hours at her grave; but his affliction was soon enhanced by the nocturnal appearance of asthmatic

[1] Phœbus, *op. cit.*, p. 30

[2] J. Ferrus, "Asthme", in *Dictionnaire*, en 30 volumes, tom. iv, 1833.

seizures. Lebert was consulted on the case, but was unable to detect anything abnormal in the organs of respiration and circulation. Here would have been all the diagnostic elements of "emotional" asthma, yet on examining the urine Lebert found it loaded with albumen and casts.[1] The dyspnœa was, therefore, a symptom of pulmonary œdema which accompanies certain forms of Bright's disease.

5. *Treatment.*—The instances in which asthma is recorded as having been cured by *gangrene of the intestines,*[2] *by fracture of the ribs,*[3] *and by sudden and strong passions,* are of too exceptional occurrence to substantiate the adopted theory, and may, therefore, at once be omitted from consideration. It is from the action of certain drugs that the nervous origin of asthma is deduced. "Of all the different kinds of evidence", Hyde Salter says, "on which we build our theories on the pathology of diseases, there is none more convincing, or that tells a plainer tale, than that which is derived from therapeutics. The success of a remedy given on certain principles proves the correctness of the principles on which it was given, and the known action of a medicine directly implies the nature of the pathological state that it relieves, as it shows

[1] *Klinik d. Brustkrankheiten*, vol. i, p. 782.

[2] Rennes, "De la disparition des accès d'asthme sous l'influence d'une affection abdominale".—*Gaz. des Hôpit.*, 1855, p. 533.

[3] Théry, *op. cit.*

that in any case of its successful administration, the pathological state must have been such as that known action would antagonise or correct. This reflected evidence has all the force of the fulfilment of a prediction, like the reappearance of Halley's comet at the exact time that its discoverer foretold."[1] "It must be admitted, however, that the remedies for asthma are of very irregular and uncertain operation; that, probably, there is no single remedy that is not inoperative in a large number of cases; that that which is useful in one is valueless in another, while there are many cases that resist all remedies."[2] If, nevertheless, the dyspnœa occasionally ceases after the exhibition of certain drugs, this amelioration is inconclusive, because "*the most blind and purposeless treatment may be attended by the happiest results*".[3] For this reason, Trousseau also derides the homœopaths who boast their successes in the treatment of this affection ;[4] yet he himself concludes that the dyspnœa, which in one case subsided after the burning of stramonium, was, therefore, nervous asthma.[5]

Only narcotics are, in general, more or less constant in their effect. But, if the relief of dyspnœa by the exhibition of these remedies were to prove the nervous origin of asthma, pneumonia, pleurisy, and tuberculosis would for the same reason be nervous affections. The

[1] *Op. cit.*, p. 183. [2] *Ibid.*, p. 3. [3] *Ibid.*, p. 104.
[4] *Op. cit.*, vol. ii, p. 446. [5] *Ibid.*, p. 444.

fact is that though narcotics *appeal* to the nervous system, inasmuch as they blunt the irritability of the centre of respiration, they simultaneously diminish also the activity of those functions and secretions which require oxygenated blood and, therefore, a free supply of air.[1] Their action may be traced even to the individual blood-corpuscle, which, under the influence of morphia, f. i., diminishes in size[2] and is partially deprived of its capacity of absorbing oxygen.[3] Biermer, however, maintains that, because chloral allays spasm, the prompt departure of the rhonchi and of the other symptoms of the asthmatic paroxysm after the administration of that drug, affords indubitable evidence of the nervous nature of the disease.[4] Yet, for aught that is known to the contrary, the presumed bronchial stricture may nevertheless persist; only the available supply of air suffices for the diminished respiratory wants of the body, while the shallow respiration is incapable of producing the stenotic sounds. Moreover, in the case reported by Leyden,[5] the effect of chloral was by no means so prompt as alleged, for though the subjective symptoms subsided, the wheezing persisted, proof that either the dyspnœa and the

[1] C. J. B. Williams, *op. cit.*, p. 47.

[2] Manassein, *Ueber die Dimensionen der rothen Blutkörperchen.* Berlin, 1872, p. 31.

[3] O. Leichtenstern, *Zeitschr. f. Biol.*, vii, 197-236.—Joh. Ranke, *Grundz. d. Physiol. d. Mensch.* 2 Aufl. Leipzig, 1872, p. 473.

[4] *Bronchial Asthma*, etc., p. 1 *seq.* [5] *Loc. cit.*, p. 335.

rhonchi do not proceed from, or chloral does not allay, the bronchial spasm.

6. *Periodicity.*—Little need be said concerning the periodicity of asthma. Its significance dates from the time when lunar influence was credited with the production of disease.[1] At present, the recurrence of the dyspnœa shows but the return of its exciting causes, from which, if unknown or unsuspected, it is inadmissible to infer an occult agency of the nervous system. An old woman, labouring under chronic bronchitis, will certainly be roused early in the morning by her "*diurnal asthma*",[2] so long as, having spent the night in a badly ventilated room, her air passages are clogged with mucus. After expectoration, which at times requires considerable effort, but which is generally assisted by the customary "cup of hot tea", the freedom of respiration is restored in such cases. Yet, notwithstanding all elaborate theories, these symptoms are in no way significant of a disturbance of innervation.

7. *Associated and Precursory Symptoms.*—Stress is laid on the copious discharge of limpid urine in the early part of, and on the unwonted hilarity or languor and drowsiness preceding, an attack of asthma; all these being supposed to point to the nervous character

[1] J. Russel Reynolds, *Epilepsy: its Symptoms and Treatment.* London, 1861, p. 147.

of the disease."[1] But abundant diuresis, because it occurs in hysteria, neuralgia, and frontal headache, is not, therefore, characteristic of nervous affections. An increase of the renal discharge is produced under a variety of conditions, too numerous, even, to refer to; it may arise from the impeded evaporation of water in the lungs during the attack; it may be a symptom, as the dyspnœa itself, of granular atrophy of the kidneys. So also the unwonted hilarity and animation have no other significance than that they are likely to tempt the patient to some indiscretion for which he subsequently has to suffer. Again, the languor and drowsiness are indicative merely of an imperfect ventilation of the lungs, in consequence of which carbonic acid accumulates in the blood and the so-called "fatiguing" substances are retained in the muscles.[2] Languor and drowsiness are observed under those conditions in which "prolonged bodily exercise is the best remedy which the asthmatic could resort to".[3] They are the precursory symptoms of the dyspnœa in those cases in which years of suffering elapse before the experience of the patient triumphs over the prejudice of dogmatism.[4] For under the baneful influence

[1] Hyde Salter, *op. cit.*, p. 29.

[2] Joh. Ranke, *op. cit.*, p. 633.

[3] Hyde Salter, *op. cit.*, p. 309.

[4] "For the last *twenty* years, in fact ever since I can recollect, my father has been running the gauntlet of medical men in England and Scotland, without any sort of benefit; so much so, that

of the prevalent theory, the rational means of relief are deferred, because it is "puzzling to explain the beneficial effect of exercise in harmony with the bronchial spasm".[1]

8. *Symptoms.*—The subjective symptoms of asthma may be dismissed as notorious for their inconclusiveness. The sensation of "tightness" across the chest[2] which all asthmatics experience during their paroxysms is not peculiar to this, but common to every other form of dyspnœa, and affords, therefore, no evidence, as alleged, of the nervous origin of the disease. Although some, and especially educated, patients declare they feel a constriction of the air-tubes and the impeded passage of the air beyond certain points,[3] there is no doubt that they have learnt, from what of the prevailing theory is known by the public, so graphically to describe their perceptions. The feeling of constriction when the thorax is distended proves a bronchial spasm as little as a similar sensation around the abdomen in flatulency proves a constriction of the intestines. Moreover, according to the teaching of

about eight years ago he gave this plan up and took the treatment into his own hands. I ought to say *legs*, perhaps; for his only curative measure is walking exercise, and the result is, that he is now comparatively free from attacks."—Case 2, Hyde Salter, *op. cit.*, p. 311.

[1] *Ibid.*, *op. cit.*, p. 310.

[2] *Ibid.*, p. 33.

[3] Romberg, *loc. cit.*

physiology, the bronchi are insensible and the vagus transmits no muscular sensations.

Of far greater significance than any of the points hitherto mentioned are the objective symptoms. Independent as these are of the will, and not liable to distortion by the reports of the patients, their correct appreciation would allow of a safe inference as to the actual pathological condition. Yet, by a capricious interpretation, this invaluable source of information has been converted into one of specious arguments in favour of a preconceived theory. It is obvious, for instance, that, if a patient be roused from his sleep by a dyspnœal attack that lasts for five minutes and terminates immediately on the expectoration of a pellet of mucus, the sputum could not possibly be due, as supposed, to an unloading of the vessels *after* the attack.[1] The mucus requires for its formation a much longer time, and must have, therefore, been present in the air-passages previous to the dyspnœa. Admitting this much, the question arises whether the obstruction of the glottis or of a bronchus by the mucus could not have been the cause of the dyspnœa, and the answer can hardly be doubtful. But, if here be any doubt, there is no room for it as regards the obstruction of the bronchi in those cases in which "the attack ceases on the expectoration of pellets, and what seem to be *casts of the air tubes*, composed of a rather tough, gela-

[1] Hyde Salter, *op. cit.*, p. 87.

tinous, transparent greyish substance"[1]. Nor is it possible that about "half a gallon of thready sputa expectorated in a very short time after the attack could be a symptom of nervous asthma".[2]

Still more erroneous is the current construction placed upon the physical signs. It is remarkable that these are variously described by different observers. It is said that during the paroxysm,

Respiration is retarded to from 9 to 7 in a minute.[3]	The frequency of respiration is augmented to from 40 to 60 and even 80 in a minute.[4]
The lungs are unusually collapsed within the thoracic cavity, and the parietes are drawn in.[5]	The chest remains distended, its walls are kept fixed in the extreme inspiratory position, such enlargement involving all the diameters of the thorax.[6]
Inspiration is difficult; expiration is short, easy and superficial.[7]	Expiration mainly is impeded, the patient labours from four to five seconds to empty his chest.[8]
The thorax sounds dull on percussion.[9]	Percussion induces a hyper-resonant note.[10]

[1] Hyde Salter, *op. cit.*, p. 337.

[2] De la Berge and Monneret, quoted by Théry, p. 212.

[3] Hyde Salter, *op. cit.*, p. 77; Biermer, *loc. cit.*, p. 9.

[4] Canstatt, *op. cit.*, vol. ii, p. 699; Ferrus, *loc. cit.*, p. 253; Wunderlich, *op. cit.*, iii, Bd. 1, 235.

[5] Williams, *op. cit.*, p. 91.

[6] Hyde Salter, *op. cit.*, p. 76.

[7] Bergson, *op. cit.*, p. 36.

[8] Biermer, *loc. cit.*, p. 9; Hyde Salter, *op. cit.*, p. 78.

[9] Williams, *op. cit.*, p. 91.

[10] Biermer, *op. cit.*, p. 10.

Physical signs so diametrically opposite to each other cannot possibly be produced by the *same* pathological process. An attempt has therefore been made to explain those differences by assuming a spasmodic and a paralytic form of asthma. But the difficulty is that the bulk of practitioners do not admit the duality of the disease; they recognise only the *spasmodic*, yet *attribute to this the physical signs supposed to be distinctive of the paralytic form.* While thus the slow and imperfect expiration is by some considered to be pathognomonic of a bronchial stricture, Henle states that just the deficiency of this act indicates paralysis of the lungs[1]; and while Biermer insists on the stenotic sounds as characteristic of spasm,[2] Kussmaul, for the self-same reasons, considers them as characteristic of paralysis[3] of the bronchial muscles.

Hence, if we admit the accuracy of the observations —and there is no reason to doubt the reports of so many trustworthy observers— the discrepancies can be accounted for only by this, that the *paroxysms of dyspnœa were produced in various ways, and under various conditions.* This conclusion seems consistent with facts. Nothing, indeed, is more evident from reference to recorded cases of asthma, than that

[1] *Op. cit.*, p. 272. [2] *Op. cit.*, p. 1.

[3] Ueber die fortschreitende Bulbär paralyse u. ihr Verhältniss z. progressiven Muskelatrophie.—Volkmann's *Sammlung*, No. 54, 1873, p. 15.

they are merely a promiscuous collection of instances of paroxysmal dyspnœa, resembling each other only in this, but widely different in every other respect. In some it is possible to *guess* the nature of the dyspnœa; others again are so imperfectly observed and reported that it is more easy to say what they are not than what they are. But in all, as shewn by the following examples selected at random, there is little doubt that the dyspnœa was *not* one of a *primary nervous origin.*

1. Wm. C., aged 25, butcher, full-faced, and with a thick upper lip, has, since his eighth year, been subject to asthmatic paroxysms. Without known cause, the disease gradually manifested itself, commencing with cough; there seemed to be, however, a certain predisposition to it, inasmuch as his *grandfather now suffers* from asthma. His father died of phthisis. The attacks are provoked by rest, especially the *Sunday's rest*, a full stomach, and lying on the *right side.* They frequently occur early on Monday morning, and are generally preceded by *tickling in the throat, nausea,* and *perspiration.* In consequence of the dyspnœa, the thorax is barrel-shaped and hyper-resonant; the sounds of the heart are indistinct".[1]

It is not easy to perceive upon what grounds the diagnosis of "nervous asthma" has been made in this case, unless it were from the grandfather's present

[1] Hyde Salter, *op. cit.*, p. 411.

dyspnœa and the very unusual provocatives of the attacks. But these conditions are not so significant; as a rule, grandfathers who have grandsons twenty-five years old suffer from asthma; yet such occurrence hardly indicates a family predisposition. Nor is it likely that the butcher's lad had generally spent his Sundays, although days of rest, in strict obedience to approved hygienic principles, so that nothing but the "rest" could have caused his dyspnœa. The facts of this case, to judge from the imperfect report, only show that there is an apparently scrofulous patient with a history of phthisis who for years had suffered from dyspnœa, which, commencing with tickling in the throat, nausea, and lying on the right side, seems to proceed from the posterior nares, the pharynx or larynx. As these parts have never been examined, it is impossible to form a definite opinion on the nature of the asthma. It seems, however, not improbable that a chronic catarrh of the larynx had given rise to the development of a small polypus, which, by a temporary engorgement, to the production of which the Sunday affords ample opportunity, obstructed the larynx. In a similar case, in which a gentleman complained of nocturnal dyspnœa when in the right lateral posture, Oppolzer diagnosed a pedunculated tumour of the left vocal cord, and his diagnosis was confirmed by post mortem examinations.[1] It

[1] *Op. cit.* p. 383.

is needless to add that in many such instances respiration is almost completely free in the intervals between the attacks. There are no signs of laryngeal stricture, nor is there harsh cough, or hoarseness. The patients only complain of an indefinite feeling of distress in the throat, or a sensation as if mucus accumulated therein, and adhered to the pharynx.[1]

II. William Burr, aged 30, an emaciated man, speaking short, with the most marked asthmatic deformity I have ever seen. He was quite well up to twelve years ago, when he met with an accident—*fell down from a loft on his back on some flagstones.* From this he did not seem to suffer at the time ; he appeared pretty well the day after the fall, went to his work, that of a groom, as usual, and did not think any more about it. A month after this, early in August, and in very hot, thundery weather, having gone to bed perfectly well, he awoke in the night about twelve o'clock, "all of a perspiration, and as if he had been running fast, and could not breathe, with a load at his chest and a wheezing at his throat". He got out of bed and sat on the side of it, and obtained a little ease, and then was able to return to bed again and go to sleep. He awoke in the morning at six o'clock as well as usual, and went to his work and thought no more about the difficulty of breathing he had experienced in the night. He had no cold at this

[1] Niemeyer-Seitz, *loc. cit.*, p. 42.

time, there was no cough, and no expectoration. A month or six weeks after this, about twelve o'clock in the day, when he was driving a horse, being in all respects in his usual state of health, he was seized with difficulty of breathing, which became so severe in twenty minutes that he was obliged to leave off his work altogether. When the dyspnœa was at its height he was suddenly seized with coughing, and found that what came up into his mouth was hot, and spitting it out, discovered it to be *blood;* the difficulty of breathing and blood-spitting lasted about three hours, and then went off together—as soon as the breath got better, the hæmoptysis ceased. The cough was very slight, the blood was about half an ounce in quantity, not pure, but mixed with mucus. The disappearance of the dyspnœa at the end of three hours was complete. He felt weak, however, and did no work for a week, and on returning to his duties found that on attempting any brisk exercise, or the laborious occupation to which he had been accustomed, his breath became short, so that he was obliged after a fortnight to give up his situation. As on former occasions, he had neither expectoration, cough, nor symptoms of cold.

He then went to a silk mill, where his work involved no exertion; on going to work, however, morning and evening, his breath would sometimes trouble him and oblige him to rest, which he generally did in a stooping position, resting his hands upon his thighs. At this

time he was able to play with other youths, and romp and run as well as ever. One night, after he had been at the mill about six months, he was seized about twelve o'clock with an attack of the same nature as the first; he got up and sat in a chair, resting his head forward in his mother's lap (who, alarmed at his condition, had come to his assistance), and in this position he went to sleep; in about an hour he was able to return to his bed, and when he got up in the morning at six o'clock, he felt no traces of his attack and went to work as usual.

He went on then for twelve months, working every day, and during the day feeling no dyspnœa, but frequently a little night and morning, obliging him to walk to and from his work slower than the rest; and then, at the usual time of the night, twelve o'clock, he was seized with an unusually severe attack, which did not go off as before, but left him with an unusual amount of dyspnœa and incapacity for exertion throughout the day. About one o'clock on this day, breathing tolerably easily while he was still, he went a short distance with a message, and, though only creeping, very much increased the difficulty of his breathing; this obliged him to stand still, and while thus resting himself he began spitting, and again found that what he expectorated was blood. This time he spat nearly a *pint;* it *poured* out *of his mouth and nose, making quite a puddle in the road,* and *he was carried home*

in a very weak state. He was unable to resume his work for *a month,* and *during this time whenever he spat there was a little blood with the mucus.* After this the blood only appeared when the breath was unusually difficult, as on violent exertion, or in the mornings, or on the occasion of an attack, and after lasting on and off in this way for two years, entirely ceased, and for the last eight or nine years he has seen none. From the time of this severe attack with hæmoptysis he never returned to his regular work at the mill, but did little odd jobs, knife and boot cleaning, etc.; his morning dyspnœa increased and became quite regular, and sometimes of an evening too; but he slept well, and had no very violent nocturnal attack for a twelvemonth. He now found food increased his dyspnœa, and often for a month together he would have a slight attack every day after dinner, lasting for two or three hours (that is, during the time that digestion was going on), so that he has sometimes gone without his dinner to avoid the attack; then, for three or six months, perhaps, he would be free from these after-dinner attacks, and then they would come on again. Sometimes they would come on after supper, but he rarely ate supper, because he found that if he did so, he was worse the next morning. Sometimes his breath would be very bad of an afternoon, and then clear up in the evening, and he would have a good night; sometimes he would be

quite well throughout the day, but bad at night; but always the same for many days and weeks together—the habitude, the diurnal rhythm, always strongly marked. For some years he went on this way by day, with occasional intense attacks at night, occurring at from six to twelve months' interval, always coming on and waking him from his first sleep, about twelve o'clock, and going off at about three or four. Sometimes the attack was not so severe as to oblige him to get out of bed, but he would turn round and kneel up in bed towards his pillow, with his hands resting on his knees, and in this way, in an hour or two, get sufficient ease to lie down. Sometimes, with leaning forward on his elbows on the bed, or on other pieces of furniture, his elbows would be quite sore and the skin rubbed off. After some years, however, now about three years ago, the attacks ceased to leave him in the morning, but lasted on through the day, at first only a little in excess of the ordinary time, going off in the forenoon, then lasting the entire day, then not leaving him at all, but keeping him up the second night; sometimes it would come on in the morning and last a day, a night and a day, then two nights and two days; then three nights and two days, and so on; and as the attacks became longer, they became more and more intense, and the intervals shorter.[1]

Such is an example of "nervous asthma, particularly

[1] Hyde Salter, *op. cit.*, p. 368 *et seq.*

remarkable for its spontaneous occurrence at the age of eighteen, without any of the ordinary causes—bronchitis, cold, or catarrhal pneumonia—and without evidence even of an asthmatic tendency; for the increasing intensity of the disease; for the hæmoptysis; for the extreme asthmatic deformity, and for the fatal termination."[1] But few will acknowledge this diagnosis, or bring themselves to believe that such serious symptoms and death were due to the doubtful asthmatic tendency, which one sultry day is said to have roused into activity! And yet it is impossible to explain the pathology of the case, as only the subjective symptoms are reported, and even these not without a strong prejudice. Nothing is mentioned of physical signs, save the "most marked asthmatic deformity". If, nevertheless, one may venture on a diagnosis, the leading facts would seem to point to an aneurism of the descending aorta. The patient falls down from a loft on his back, on some flagstones; shortly afterwards he has an attack of dyspnœa, then hæmoptysis commences and lasts with varying intensity for a length of time; the dyspnœa gradually becomes permanent, and concurrently with it, developes the deformity of the thorax. There are cases on record where a fall from a height has caused sudden death by rupture of a *healthy* aorta, or the formation of an aneurism[2] which

[1] Hyde Salter, *op. cit.*, p. 370 *et seq.*

[2] Bailly, Arnold, and Gaujot; quoted by Luton in Article,

ended fatally after many years.[1] It is known, also, that such cases have been admitted into hospitals with the diagnosis of "nervous asthma", whereas the nature of this was revealed only by *post mortem* examination.[2]

III. A woman, sixty years of age, had for a long time suffered from pain in the abdomen and from asthma. In the spring of 1847, while busy in the field, she was seized with such intense dyspnœa, that she had to be carried home. She was greatly agitated, throwing herself about in bed, and very thirsty; her face had an anxious expression, the injected eyes were prominent and rolling, the skin was dry and hot, and the pulse was small and frequent. The percussion note was normal, but on auscultation it was found that the bronchi were filled with mucus. The administration of an emetic

"Aorte".—*Nouveau Diction. d. Med. et d. Chirurg.-pratiques*, tom. ii, p. 720.

[1] "From fifteen to twenty years."—Lebert, *Krankh. d. Blut. u. Lymphg.*, p. 396.

[2] "Private G. Parking, æt. 36, was admitted into the hospital, Arbour Hill, Dublin, on August 18th, suffering from *asthma*, and died December 2nd, 1870. Two years previously, whilst exercising a young horse in the Riding School, the animal reared and fell over with the rider underneath, the fall shaking the man a great deal; but, so far as could be ascertained, the man was not injured, and in a few days resumed his duty. On admission, there was absolutely no sign indicative of an aneurism, which, on *post mortem* examination, was found to occupy the descending aorta."—J. S. Charteres, *Dublin Quarterly Journal of Medical Science*, 1871, p. 230.

procured her great relief. On the third, fifth, and seventh days, there were fresh attacks of dyspnœa, which were ushered in by rigors, followed by heat. On the third day the lungs *became consolidated*, and on auscultation *bronchial breathing was heard* ; the cough was troublesome and the sputa contained blood ; there was also pain in the side. The bronchial breathing then gave way to mucous rhonchi, the fever and the pain subsided, and within three weeks she completely recovered.[1]

It is needless to comment on this case of "nervous asthma."

B.—*Is asthma a bronchial spasm?* Now that the grounds, upon which a bronchial spasm is assumed to be the proximate cause of the dyspnœal paroxysms, have to a great extent been removed by the preceding discussion, it would be as tedious as it is superfluous, to recur to arguments based on mistaken facts. It suffices, in order to refute the present theory, to show that a spasmodic stricture of the bronchi is not merely inconsistent with the clinical aspect of the disease, but actually impossible.

Histological research has fully established the existence of an almost continuous layer of muscles between the external and the internal fibrous coats of the bronchi. In the tubes of not less than one millimetre in

[1] Bergson, *op. cit.*, p. 66.

diameter, the contractile tissue constitutes about one-fortieth of the thickness of their walls; but in those of smaller calibre, the muscular fibres become separated from each other and are gradually diminished in number until they dwindle down to one single stratum.[1] Towards the infundibula, the fibres are said again to converge, so as to form a kind of sphincter[2]—replacing, as it were, the cartilaginous plates at the corresponding portions of the larger bronchi.

It does not appear, however, that the bronchial muscles are actively engaged in the mechanism of respiration, because their contractions are for this too slow, and also too feeble.[3] Their function seems to consist merely in opposing the traction to which the air tubes are continually subject, just as the vascular muscles serve to resist the dilating force of the blood current.[4] But a spasm of the bronchial fibres so as to produce dyspnœa, is, notwithstanding their liability to it, a matter of unwarranted assumption. The experiments from which that theory has been deduced, disprove rather than support that inference. The bronchial contractility has been observed only in lungs that were either removed from the body, or detached from their connections with the pleura. Yet

[1] F. E. Schultze in Stricker's *Handb. d. Lehre von den Geweben.* Leipzig, 1870, p. 468 *et seq.*

[2] Rindfleisch, *loc. cit.*

[3] Paul Bert, *op. cit.*, p. 380 *et seq.*

[4] Biermer, *Bronchialasthma*, p. 4.

in the *closed* thorax there are widely different conditions; here the lungs are distended beyond their normal volume, which from after the first inspiration until after the last expiration, they, in vain, endeavour to resume; and the extent of the distension may be judged of by comparing the cavity of the opened chest, and the size *of the collapsed* lungs. *This distension, however, proves an insuperable impediment to the contractions of the bronchi.* In his experiments, Dr. C. J. B. Williams found that, when the lungs were inflated, electrical irritation produced *no* effect; only after a certain volume of air had been expelled from the lungs, did the bronchi exhibit signs of contractility, which even now was less pronounced than when the organ was collapsed.[1] The same observation was made by Paul Bert; he discovered that the failure of his numerous experiments was attributable alone to the *inflation* of the lungs.[2] Hence, as this obstacle exists normally, and in a still greater degree in emphysema, with which, by necessity or by accident, asthma is almost always associated, there can consequently be *no* bronchial stricture.—Moreover, the clinical features of the asthmatic paroxysms afford abundant evidence against that theory. Apart from the improbability that muscles could remain uninterruptedly contracted for

[1] *Op. cit.*, p. 322.

[2] "J'insufflais trop les poumons dont je voulais enregistrer les mouvements."—*Op. cit.*, p. 375.

days[1] and weeks together, there are the physical signs during an attack, which are quite inconsistent with the assumption of a bronchial spasm. For :—

1. If the bronchial muscles were engaged in respiration, they could be only auxiliaries of expiration. Their increased activity, while greatly impeding the admission of air, would on the contrary facilitate its expulsion, and each such act, now performed with increased energy and without any obstacle, ought to be rapid and complete, comparable, perhaps, to the closure of the pulmonary valves in cases of mitral disease. In reality, however, the opposite takes place, inspiration is relatively easy, whereas expiration is laborious and ineffectual.

2. If the bronchial spasm *oppose* expiration there would still be means available, sufficient to overcome the obstacle and to *prevent* the occurrence of the dyspnœa. The vital tonus of the lungs amounts to only from one fourth[2] to one fifth[3] of their contractile energy, and experiments have shewn that though the bronchi may be contracted, the elasticity of the air-vesicles is yet able to expel the air.[4] It is therefore hardly conceivable that those circular fibres should, by

[1] Hyde Salter knew a patient who stood grasping the back of a chair for two days and one night, unable to move.—*Op. cit.*, p. 74.

[2] Donders' *Zeitsch. f. ration. Mediz.*, Bd. iii, H. 3.

[3] Wintüch., *loc. cit.*, p. 197.

[4] *Ibid.*, p. 200.

the spasm, gain so much in strength as to be capable of resisting the united forces of (*a*) the elasticity of the fibrous tissue, increased, as it is, by the tension during the previous inspiration; (*b*) the contractions of the auxiliary muscles of expiration; (*c*) the external atmospheric pressure; (*d*) the resiliency of the chest walls, and (*e*) the tendency of the compressed intestinal gases to expand. The opinion of Biermer that this forcible expiration compresses, instead of opening the bronchioles,[1] is quite unwarranted. Not to speak even of the defiance of Breuer's law as involved in that supposition, it is evident that the expiration could not have the alleged effect; for all its auxiliary forces, such as the resiliency of the chest walls, and the atmospheric pressure, would act first upon the peripheral, before they could reach the more central portions.

3. Again, if the bronchial spasm oppose both phases of respiration,[2] it would be quite unintelligible that inspiration should succeed and expiration should fail in attaining their objects. That the former actually succeeds in admitting air into the lungs is shewn by the emphysematous distension during the paroxysm. But as the force of the expiratory pressure is normally greater than that of the inspiratory traction, and as this proportion remains here also constant, the obstacle

[1] *Conf.*, p. 44.

[2] Biermer, *loc. cit.*, p. 5.

being, as assumed, equal in both phases, it is evident that what the smaller force *can*, the greater *must*, effect.

4. Spasm of the bronchi would produce collapse of the lungs, so that the intercostal spaces would be drawn in and the diaphragm would be raised. In point of fact, however, the organ is, during the paroxysm, distended to its utmost limits in all directions. To say that the spasm is confined to the bronchi and does not reach the infundibula, would not explain the reason why it should stop short at the comparatively thick muscles with which these are provided, as stated above. But if the pleural traction suffice to cause the emphysematous distension of the alveoli, the bronchi could not be constricted during the permanent inspiratory position of the chest. For there is a direct continuity between the subpleural, alveolar, and peribronchial tissues, so that the force exerted upon the air-vesicles would be equally transmitted to the adventitious sheath of the air-tubes.

5. The respiratory murmur is said to be inaudible during the attack, "because the conditions of its production do not exist; sufficient air is not admitted to generate it".[1] But if the bronchi were so completely constricted as alleged, life could not be sustained for any length of time. Nor is it possible that the feeble tide of air should yet produce sonorous and

[1] Hyde Salter, *op. cit.*, p. 80.

sibilant rhonchi, often of such intensity as to be heard even at a distance.

6. The great mobility of the rhonchi is incompatible with the assumption of a bronchial spasm. The respiratory movements are uniformly effected throughout the entire organ, and no peristaltic action of the bronchi is known to occur. Therefore, if the rhonchi cease at one spot and reappear at another, the *points of stricture* cannot be produced by spasm.[1] Nor is it possible that the nervous irritation would be successively transmitted to different portions of the same muscular organ. Notwithstanding Hyde Salter's assertion that expiration favours bronchial spasm,[2] there are the observations of Laennec, Williams, and Walshe,[3] that loud reading and speaking during the fit, *i.e.*, prolonged expiratory acts, cause, on the contrary, the rhonchi to disappear and give rise again to the previously suppressed respiratory murmur. How expiration could "break the spasm", is beyond comprehension; and, as the fact is indubitable, this interpretation must be erroneous.

But in the vast majority of instances the bronchial muscles are so greatly impaired in nutrition as to be incapable even of contracting. Cases of pure and idiopathic asthma—in which the lungs are, to all appearances, perfectly sound,—are admittedly of very

[1] Hyde Salter, *op. cit.*, p. 80.

[2] *Op. cit.*, p. 53.

[3] *Diseases of the Lungs*, etc., p. 548.

rare occurrence ; and if they occur at all, they are, after a short persistence, accompanied by bronchitis and emphysema. For the present, it is quite immaterial what relation the textural changes hold to the asthmatic paroxysms ; it suffices to state the fact that they do simultaneously exist. Now, in the acute bronchitis, the submucous and muscular layers of the tubes are the seat of the collateral œdema, of which the consequence is an *acute relaxation* of the bronchial walls—a process analogous to the one that produces paralysis of the intestines in cases of peritonitis. It is true that this inflammatory dilatation may completely subside with the cessation of the acute disease. But when the attacks are repeated, "then the capillaries and venules, long distended, never completely recover themselves, their tone is lost, and the pulmonary congestion, manifested by chronic dyspnœa and expectoration, is permanent. This pulmonary congestion, involving, as it does, the bronchial tubes, and occluding them with mucus, becomes, in its turn, a source of bronchial irritation, and then tends to excite and keep up the asthma which has caused it".[1] That this dyspnœa, however, cannot be a spasm is evident from the histological details of the process. The pathological changes primarily consist in an abundant infiltration of the bronchial walls with white blood-corpuscles, which, in the course of their development or dis-

[1] Hyde Salter, *op. cit.*, p. 323.

integration, are either converted into connective tissue or undergo fatty degeneration. Thus the muscular fibres of the air-tubes are paralysed by the degenerative changes that take place around them. Therefore, when the dyspnœa has lasted for some time, the *dilatation* of the air-tubes becomes permanent, and, indeed, the most dilated bronchi Hyde Salter has ever seen, was in a case of "*spasmodic* asthma".[1] It is superfluous to add that under these conditions the contractility of the bronchial muscles is totally destroyed. The same loss of contractility takes place in emphysema, because the smaller bronchi participate in all the nutritive changes of the alveoli;[2] while the larger tubes are the seat of chronic inflammation or frequently also of dilatation.[3]

[1] *Op. cit.*, p. 157.

[2] Förster, *Handb. d. patholog. Anatomie.* 2 Aufl. Leipzig, 1863, p. 300.

[3] Rokitansky, *op. cit.*, vol. iii, p. 50 *et seq.*

CHAPTER IV.

DEFINITION, PATHOGENY, AND ETIOLOGY.

A. *Definition and Pathogeny.*

THE intense and intermittent dyspnœa which, mainly on account of its fitful appearance and departure, and its seeming independence of structural lesions, has been hitherto attributed to a derangement of innervation, is in reality due to pathological changes of the lungs. It is a symptom, and as such accompanies the idiopathic forms of chronic bronchitis and emphysema, as well as those that complicate the various cardiac and pulmonary affections. The dyspnœa in connection with the latter evinces, in an almost unmistakeable manner, its symptomatic character, so that only erroneous views as to the causes of its intermittency, have led to the supposition of its primary nervous origin. As those views have been fully discussed in the preceding pages, no further reference will be made to the numerous varieties of the symptomatic asthma described by writers. The case, however, is different with regard to the dyspnœal paroxysms that accompany the idiopathic forms of chronic bronchitis and emphysema. The anatomical changes peculiar to these affections are not only slow and

latent in their progress, but even after their complete development they are so imperfectly accessible to diagnosis as to appear quite out of proportion to the intensity of their symptoms. Those asthmatic seizures are therefore always the most conspicuous, after a time even the sole, indication of the pathological conditions that take place in the lungs; and hence they possess all the clinical importance of a substantive disease, and require a detailed account of their nature and origin.

That asthma is a mere symptom, can readily be proved. The recognition of this important fact has greatly been impeded by the exclusive attention paid to the clinical features of the paroxysms, and by the persistent attempts to deduce from them alone the nature of the dyspnœa. There are, however, but few chronic affections which, at a given period of their existence, present symptoms in themselves so characteristic as to admit a safe inference as to their origin. Even in those cases where the morbid phenomena are directly traceable to organic lesions, it is requisite to refer to the history of the disease in order to recognise its pathology. No valid reason can, therefore, be adduced why this established mode of procedure should be departed from in respect to asthma; on the contrary, its adoption is the more necessary, since objective examination, which generally affords the most important information, proves here of no avail. Nor is

it admissible to disregard altogether those circumstances which, although separated by time, yet precede and follow the dyspnœal seizures with a striking regularity. Insignificant as they may be singly, those antecedents and sequelæ throw light on each other by their mutual relation, and exhibit, as it were, the life-history of asthma.

1. *Constitution.*—It is a striking fact, that constitutional defects appear to be invariably associated with asthma. The very appearance of the patients is characteristic in that respect. The great majority of them are stunted or ill grown, while their pale and sallow skin, their atrophic muscles, and their dilated subcutaneous veins, betray a state of general malnutrition. Moreover, the square and prominent forehead, the deformities of the spine, and the disproportionately large or abnormally narrow thorax,—all these are evident traces of bygone rickets. Others, again, may, at first sight, be taken for healthy and robust subjects; yet many of them owe this favourable opinion to their tendency to obesity at an early period of life—a sign, if not of rickets in a special form, at any rate one of premature senescence. Now, experience has warranted the assumption that the nutrition of the internal organs corresponds to that of the bony and muscular systems, and that in those individuals constituted as just described, the lungs, above all, are deficient in power of resistance to, and of repair after, injuries.

2. *Previous Disease.*—With the exception of the comparatively rare instances in which the dyspnœa is produced by a foreign body in the air-passages, asthma generally gives fair warning of its approach. Those subject to the disease have, previous to the appearance of the typical paroxysms, suffered from an acute or chronic inflammation of the respiratory organ. Of the 223 cases collected by Hyde Salter, the original cause of asthma was not ascertained in 40; in the remaining 183 cases, the original causes are stated as follows:—

Chronic catarrh, colds, and catarrhal pneumonia complicating whooping-cough, measles, and typhoid fever	132
Apparently spontaneous	10
Inherited and constitutional	11
Climate and miasms	8
Hard work, anxiety, and over-exertion	6
Outbreak and disappearance of eczema	5
Small-pox driven in on the lungs	1
Liver complaint	1
Bilious fever	1
Weak stomach	1
Indigestion	1
Supper of meat, onions, and hard beer *(sic)*	1
Sudden soberness	1
Hay-making	1
Suffered in teething	1
Sudden cessation of menses	1
Running violently	1
Total	183

From these statistics, it appears that bronchitis and catarrhal pneumonia have preceded the dyspnœal seizures in eighty per cent. of all cases; and this proportion would be still greater, if it were permitted to exclude those etiological factors, which seem, as such, highly improbable. It is needless to add, that this great frequency is not mere matter of accident, but points to a certain relation between the events. What influence bronchitis and pneumonia have upon the origin of asthma, will be evident from their anatomical details. In sixty-two per cent. of those cases the inflammation occurred in the first decade of life, and was either idiopathic or a complication of whooping-cough and measles. Now, in feeble, narrow-chested children, and in those affected with thoracic deformities more especially,—and these supply the greatest contingent of asthmatics,— bronchitis, though commencing in the larger, has the tendency to spread to, and even beyond, the smaller tubes. Under these circumstances, the bronchial wall is inundated with a gelatinous (synovia-like, Rokitansky) substance, consisting of serum and white blood-corpuscles. The same kind of exudation exists also in the interlobular tissue, and here diffuses itself so as to reach even the pleura. Some of the alveoli are inflamed; some are filled with aspired mucus; others are in a state of atelectasis; and, again, others are the seat of a vicarious emphysema.[1] It

[1] Ziemssen, *Pleuritis u. Pneumonie im Kindesalter.* Berlin, 1862, p. 293 *et seq.*—Buhl, *loc. cit.*, p. 112.

is true, these anatomical details are derived from fatal cases, and it is quite possible that they may differ in extent and intensity in the surviving cases. The difference may be such as to allow a complete resolution, and with it a complete recovery, although, to all appearances, the constitutional disposition of the patients is by no means conducive to that favourable issue. Again, "colds" and bronchitis in the adult are elastic terms for very variable anatomical conditions. If a patient be affected with a slight cough, and if, what rarely happens, he should seek advice for it, the normal resonance of the thorax, and the presence or absence of "catarrhal" rhonchi, by no means prove that the inflammation is limited to the internal mucous layer of the bronchial lining. The subsequent course of innumerable cases of this kind plainly shows that the process commenced in the interlobular and peribronchial tissue, and thence gradually spread to the internal surface of the bronchi; yet, as that process neither diminishes the volume of air in the lungs, nor impedes its entrance and exit to and from them, the full extent of the disease cannot be recognised at its early stages. However this may be, there can be little doubt that if an individual—child or adult, who, up to a certain time enjoyed good health—be attacked with what seems to be a bronchial affection; that from this he apparently recovers, but remains henceforward subject to asthma; all this strongly argues that the respira-

tory organ has been permanently injured by the preceding inflammation. There yet remains a minority of instances in which asthma is apparently congenital or spontaneous. But the question is, how far, on the one hand, congenital atelectasis, and how far, on the other, those insidious interstitial changes of the pulmonary tissue, are responsible for it. There is no means of determining when and how commences the process that ultimately leads to granular atrophy of the kidneys. And, undoubtedly, a closely analogous process takes place in the lungs. Moreover, many patients, suffering from what is called chronic bronchitis, are by no means precise in their statements as to its origin. They date the first commencement of their illness from their recent catarrhal attack, which has only aggravated their condition; ignoring altogether the cough, expectoration, and shortness of breath, to which they have gradually become inured during the slow development of their disease.[1]

3. *Sequelæ.* Emphysema and bronchiectasis, the one preponderating over the other, as the case may be, soon appear in the train of asthma. The fact that a nutritive and a mechanical element concur to their production, deprives the theory that these affections are consequences of asthma, of all foundation. Eppin-

[1] Headlam Greenhow, *On Chronic Bronchitis*, etc. London, 1869. p. 28.

ger[1] denies the nutritive disturbance of the pulmonary tissue, and endeavours to show that severe cough is the sole and exclusive cause of emphysema. Without entering, however, into a discussion of his view, it is enough to say, the origin and persistence of a severe cough in *perfectly healthy lungs*, would be quite inconceivable. So far as asthma is concerned, there is evidence, already alluded to, that a predisposition inherent in the lungs is mainly instrumental in the causation of alveolar and bronchial dilatations. That predisposition, however, is apt to be overlooked; the changes peculiar to it pursue their course gradually and unperceived, and their existence is known only when their destructive work is completed.

Hence the antecedents and sequelæ of asthma manifest themselves by their nature, as *one continuous though protracted pathological process. Asthma, therefore, is only one link in a chain of quasi-independent affections, which commences with inflammatory changes of the pulmonary tissue, and terminates with emphysema or bronchiectasis.*

Thus, according to this definition, "the essential or idiopathic asthma" of writers corresponds to the dyspnœa, accompanying the gradual and latent progress, and their "symptomatic or catarrhal", to the fully developed state of chronic bronchitis and emphysema.

[1] *Das Emphysm der Lungen. Prager Vierteljahrsch. f. Heilkunde*, Band iv. 1876.

The various classifications of the disease that have been proposed are, for obvious reasons, inapplicable to the present case.

In view of the different sense in which asthma is here taken, the question arises whether it be advisable to retain the term with which the idea of a nervous disturbance has at all times been associated? There is, indeed, a certain suspicion that misconceptions as to the nature of those paroxysms may easily arise in this way. Yet asthma well expresses the clinical peculiarities of that form of dyspnœa, and may therefore be advantageously adopted for the purpose of description, without prejudice, however, to its pathology.

B. *Etiology.*

I. Predisposing Causes.

Like most other morbid phenomena, asthma also depends upon the concurrence of predisposing and exciting causes. Of these etiological elements, the former are decidedly the more important. They alone are capable of explaining why, of two individuals who expose themselves to the same injurious influence, the one escapes unhurt, whereas the other is seized with dyspnœa. The present inquiry has shewn that the tendency to asthma is produced by definite organic lesions of the respiratory organs; only the imperfect means of diagnosis and the rarely fatal issue of the disease, at the time when

its clinical features are most characteristic, are the cause that the existence of those lesions has been so constantly overlooked. Still, the known pathology of the antecedents and sequelæ permit the safe inference that the stage intermediate between them also corresponds to certain textural changes.

1. *Anatomical Lesions.* — The anatomical details vary in extent and intensity. A copious exudation of serum and white blood-corpuscles is already stated to be the starting-point of the affection. Constitutional and local causes determine the subsequent fate of that exudation. Of the former, little is definitely known; only this much may be supposed, that the blood must be deprived of some of its essential properties on account of the great loss of its constituents, which, after their complete development, are destined to perform the most important functions, but whose regeneration is retarded owing to the existing febrile process. As regards the local causes, there is reason to believe that the crowding of the leucocytes in the bronchial and interstitial tissue not only impedes their development, but leads to compression and obliteration of the adjoining capillaries. An extensive thrombosis of the capillaries,[1] whether from stasis, compression,[2] or endarteritis,[3] thus diminishes the

[1] Isaaksohn, *loc. cit.*

[2] Friedländer, *Untersuchungen über Lungenentzündung*, etc. Berlin, 1873, p. 22.

[3] Köster, *Endarteriitis u. Arteriitis. Verhandl. d. Natur-*

supply of arterial blood to the affected parts. The white blood-corpuscles are softened and reabsorbed. This process of liquefaction implicates, at the same time, the tissues in which it takes place. The bronchial muscles, paralysed by the preceding inflammation, now undergo fatty degeneration;[1] while the elastic fibres become hyaline and brittle, each fragment being composed of round or oval granules.[2] Atrophy of the contractile and elastic elements of the lungs, is, then, the result of those degenerative changes. As the pleural tension, however, remains the same, while the resistance of the atrophic lungs to the excentric traction is diminished, the bronchioles and alveoli are consequently dilated. This atrophous dilatation[3] is either cylindrical or saccular, and is most marked in the bronchioles of the fourth subdivision, extending hence to the alveoli, so that the dilatation of the one occasionally preponderates over the other. Such lungs, inflated and dried, present the aspect of a large-holed sponge, the cavities varying in size from that of a hemp-seed to that of a pullet's egg.

Not unfrequently the white blood-corpuscles are, for various reasons, endowed with sufficient vitality, so as

histor. Vereins zu Bonn. Sitzungsb. December 20th, 1875, page 320.

[1] Buhl, *op. cit.*, p. 22.

[2] Cornil, "Altération des fibres élastiques du poumon, etc." —*L'Union Médicale.* No. 81. 1874.

to develope themselves into permanent tissue. An active hyperplasia and hypertrophy are then observable in the parenchymatous stroma of the lungs. This genuine peribronchitis and intestitial pneumonia also lead to obliteration of capillaries and air-vesicles by the firm connective tissue, the fibres of which radiate in all directions; while the contractile and secreting structures of the bronchi are either destroyed[1] or greatly impaired in their functions and nutrition. Here, again, the tonus and elasticity of the lungs are so greatly diminished, that the pleural tension readily effects the "hypertrophous bronchiectasis"[2] and the vicarious emphysema.

Unless the volume of the blood decreases in proportion to the reduction of the respiratory surface, it is evident that the pervious vessels of the lungs must be in a state of vicarious distension. But the destruction of the pulmonary capillaries generally takes place at the time when development is most active, and when, with the growth of the osseous system more particularly, the volume of the blood also increases. Hence, the greater amount of work which the right side of the heart is now called upon to perform, in order to force a relatively larger quantity of blood through the contracted lungs, produces hypertrophy

[1] R. H. Fitz. Virchow's *Archiv*, 1870, Bd. li, page 126 *et seq.*

[2] Buhl, *op. cit.*, p. 56 *et seq.*

of the organ,—a consequence to which Dr. Peacock[1] was the first to call attention. Under the influence of this increased pressure, the pulmonary vessels, unprovided as they are with a vascular tonus,[2] readily dilate ; and, in order to compensate for the loss of the capillaries, extensive anastomoses form between the pulmonary artery, on the one hand, and the bronchial and pulmonary veins on the other, by means of vascular arches, remarkable on account of their great length, their equable diameter, and their want of branches.[3] The surface of the bronchial mucous membrane then appears studded with granular or villous elevations ; on microscopic examination, these are found to be vascular papillæ, pyramidal or club-shaped, consisting of a tortuous vessel, elevated over the surface and surrounded with connective tissue.[4]

The inundation of the pulmonary parenchyma with nutritive fluid, causes enlargement of the bronchial glands, which, as a rule, participate in all affections of

[1] *Monthly Journal of Medical Science.* Edinburgh, 1854, vol. xix, p. 403 *et seq.*

[2] E. Badoud, "Ueber d. Einfluss d. Hirns aufd. Druck in d. Lungenarterie."—*Verhandl. d. Würzb. physik-mediz-Gesellschaft*, vol. viii, Heft 1 and 2. 1874.

[3] Rindfleisch, *Lehrbuch d. patholog. Gewebelehre.* Leipzig, 1871, pp. 347, 348.

[4] Carswell, Reynaud, and Virchow, quoted by Biermer, *Krankheiten d. Bronchien*, p. 661.—Villemin, *loc. cit.*

either the surface or substance of the lungs. In many instances, that enlargement dates from the whooping-cough and measles, and diminishes or increases with the diminution or increase of the quantity of, as well as the obstacles to, the lymphatic current. Other changes in the bronchial glands, beyond the mere hypertrophy, are mainly determined by the constitutional proclivities of the patients.

So long as the energy of the heart remains unimpaired, the organism is in no perceptible manner affected by the reduction of the respiratory surface. But when the cardiac muscle degenerates, as it necessarily does, in consequence of the overstrain, a general dilatation of the venous system manifests itself. Varicose veins of the lower extremities, hæmorrhoids, varicocele, and dilatation of the ovarian and uterine plexus,[1] are almost constant occurrences in asthma. Less frequent, but by no means rare, is the enlargement of the thyroid gland,[2] which apparently comports itself towards the lungs as the spleen does towards the liver in cases of hepatic cirrhosis. Occasionally, under the influence of the increased venous pressure,[3] a kind of erectile tissue forms upon the turbinated

[1] Ferber, "Niesekrampf."—*Archiv d. Heilkunde*, vol. x.

[2] Lebert, *Handbuch d. prakt. Medicin.* 4 Aufl. 1871, Bd. ii, p. 26.

[3] Albert Thierfelder, *Pathologische Histologie.* Lief i, 1872, plate 1.

bones,[1] and naso-pharyngeal polypi develope themselves, which Voltolini[2] erroneously regards as causes of asthma.

According to the observations of Verneuil[3] and the experiments of Ranvier,[4] the varicose dilatation of a vein always commences at its radicles. As the healthy nutrition and function of an organ depend, to a great extent, upon the integrity of its capillaries, it is readily conceivable that profound changes of the surrounding tissues are liable to occur and actually take place in consequence of that dilatation; for the increased pressure in, and the thinning of, the venous radicles, favour the escape of serum and white blood-corpuscles.[5] The various "catarrhal" affections of the external and internal linings of the body,—the skin and mucous membranes,—are thus the consequence of, and in their turn increase the tendency to, asthma.

2. *Diathetic Disorders.*—The connection between skin-diseases and gout on the one hand, and asthma on the other, has been somewhat exaggerated and also perverted. The theory of Duclos and Trousseau, ac-

[1] Kohlrausch, "Ueber d. Schwellgewebe an den Muscheln d. Nasenschleimhaut.—Müller's *Archiv*, 1853, p. 149.

[2] *Die Anwendung d. Galvano-Kaustik*, etc. 4 Aufl. Wien, 1872, p. 246.

[3] Lebert, in Virchow's *Handb.* Bd. V, Abth. II, Lief ii, p. 547.

[4] *Archives de Physiologie.* Série II, tome i, 1874, p. 456 *et seq.*

[5] Billroth, *Die allg. chirurg. Pathologie u. Therapie.* 4 Aufl. Berlin. 1869. p. 584.

cording to which a "herpetic principle" in the blood attacks in a fitful manner, at one time the skin, at another the bronchial mucous membrane, is devoid of all positive proof. All the reported cases, upon which that theory relies, are certainly not conclusive in its favour. A house-painter suffers in 1859 from eczema, which completely subsides; but in 1861 he has a dyspnœal seizure. Moutard Martin observed a case in which an eczematous eruption was followed, at an interval of *six years*, "by asthma".[1] What influence the cutaneous affection could have had in these cases upon the appearance of the dyspnœal seizures, is beyond comprehension. If, nevertheless, skin-diseases and asthma are frequently associated, the reason of that occurrence must be sought in the nutritive changes, which, by the dilated venous radicles are produced in the external integument. "Eczema" of the lower extremities generally accompanies the varicose dilatation of their veins;[2] but this vesicular eruption is only an indication of the facility with which serum and white blood-corpuscles escape through the altered vascular walls. Under these circumstances, the causes productive of cutaneous affections find a very favourable soil for the display of their effect in the asthmatic. And, as extensive congestion of the skin would necessarily produce a collateral anæmia of the lungs, in the same

manner as, but much more effectually than, a sinapism, —judging from the extent of the surface implicated,— the alternation between asthma and a dermatosis can be readily accounted for.

A similar interpretation may, perhaps, be placed upon the association of gout and asthma. Both seem to be the co-ordinate results of the same constitutional cause; for an absolute or relative plethora, such as it undoubtedly exists in both diseases, is sufficient to explain the origin of the organic lesions of the lungs, as well as that of the articular affections. As regards the supposed retrocession of gout to the lungs, there is, as yet, no fully ascertained case in which its characteristic deposit has been found in that organ. But if lithic acid were to accumulate in the lungs, there is little doubt that it would here produce the same effect as it does in the joints.

3. *Inheritance.*—A hereditary element seems to be influential in the production of the asthmatic tendency. About forty per cent. of the patients state that some members of their families had been subject to the disease. Yet, in eleven out of seventeen cases, that hereditary predisposition is traceable to grandfathers and grandmothers.[1] Considering, however, the vague application of the term asthma, and the fact that atrophic emphysema, with its intermittent dyspnœa, is almost a physiological attribute of old age, no great reliance can

[1] Hyde Salter, *op. cit.*, p. 116.

be placed upon those statements. This much, however, is certain, that feeble parents produce a feeble offspring, and that just as physiognomy and mental disposition are transmitted, so a peculiar weakness of texture and a deficient power of resistance to, and of repair after, injury, may be communicated from one to another generation. Where the paroxysms appear soon after birth, it does not follow that, as Hyde Salter suggests,[1] the disease is congenital. A premature respiration of the fœtus on its passage through the maternal canal may, by the aspiration of fluid, lead to atelectasis or bronchitis.

4. *Age.*—The first ten years of life are most obnoxious to asthma, and the reason is, that infantile bronchitis and catarrhal pneumonia, complicating measles and whooping-cough, are most frequent at that time. But, though the foundation of the disease may be laid at so early a period, its manifestation depends upon the extent and intensity of the primary affection. In comparatively rare instances, the predisposition slowly develops, and declares itself only in youth and early manhood. At a more advanced period of life, the involution of the lungs, premature or in connection with that of the body in general, constitutes a very marked liability to the dyspnœal paroxysms. In old age, however, asthma soon loses its peculiar characteristics, inasmuch as the intermittent is converted

[1] *Op. cit.*, p. 112.

into a permanent dyspnœa, while the anatomical lesions, upon which this depends, are readily discernible.

5. *Sex.* — Asthma is decidedly more prevalent among men than women. How far the wear and tear of the body, proceeding from habits and occupation, are accountable for its greater prevalence in the male sex, is not quite known. The supposition that those suffer most who most expose themselves to the inclemencies of the weather, as, for instance, "costermongers, cabmen, and Covent Garden porters",[1] is not borne out by facts. On the contrary, those who lead a sedentary and indoor life, supply the greatest contingent of asthmatics. The occupation, however, can have no bearing upon the question in the first ten years of life, where that preponderance of the male sex is already noticeable. It appears that the greater frequency of asthma in the latter is mainly due to the greater mortality of the female from whooping-cough.[2]

6. *Occupation.*—According to Hyde Salter's statistics,[3] about seven-ninths of the male asthmatics are gentlemen, and more than three-fourths of the female asthmatics are ladies. Thus the upper-middle and upper classes would seem to contribute more than three times as many cases of asthma as the lower.

[1] Hyde Salter, *op. cit.*, p. 403.

[2] Edw. Smith, *Med.-Chirurg. Transact.*, vol. 37. 1854.

[3] *Op. cit.*, p. 402.

Salter suggests that the greater frequency of the disease among the better classes may be attributable to the promptitude with which they bring their suffering under medical cognizance. Much, however, is due to the medical and sanitary advantages which the children of the rich possess. They are thus enabled to pull through the catarrhal pneumonia that lays the foundation of their disease, and to survive as asthmatics; whereas the children of the poor, when suffering from that affection, are cut off at once.[1] Statistics of those who seek advice at hospitals are not at hand. This much, however, is certain, that chronic bronchitis, with dyspnœal paroxysms, which among the well-to-do is generally called spasmodic asthma, is by no means of rare occurrence among the poor. Still, it appears as if the children of the rich were more liable to catarrhal pneumonia than those of the poor; for, while the latter pass, ill-clad and exposed to the inclemencies of the weather, through their whooping-cough, in the streets, with no other inconvenience than the temporary interruption of their play by a fit of coughing, the former are confined to rooms, and are anxiously guarded against the access of air—the much-dreaded draught. The observations of Bartels[2] tend to show that in badly ventilated rooms inflammation of the bronchi rapidly

[1] *Op. cit.*, p. 402.

[2] "Catarrh. Pneumonie."—Virchow's *Archiv*, vol. xxi.

spreads to the pulmonary tissue. And Geigel[1] also states that, while the most neglected children suffer mainly from derangement of digestion, those better cared for are chiefly subject to pulmonary affections.

II. Immediate Causes.

The organic lesions previously described as forming the predisposition to asthma, are, it is needless to add, by themselves incapable of producing dyspnœa. Notwithstanding the textural changes of the lungs, respiration continues to be undisturbed, and this perfect freedom of one of the most important functions, is mainly due to the beneficent activity of compensatory forces which here, as elsewhere, tend to the preservation of health, even under the most disadvantageous circumstances. Indeed, it may be said that the disease which inflicts the irreparable injury carries with it also the means of relief. However considerable the reduction of the breathing surface may be, such respiratory arrears as might be expected to result from it do not exist. They are, on the contrary, completely balanced, partly by the greater volume of the blood which the hypertrophied heart propels through the pervious capillaries, and partly by the supplementary interchange of gases that takes place in the dilated vessels on the bronchial

[1] *Deutsche Vierteljahrschrift f. öffentliche Gesundheitspflage*, vol. iii. p. 520.

surface, where a delicate epithelium hardly opposes an obstacle to that process. Even when the failing action of the heart frustates to a great extent the present arrangement, there are nevertheless means by which respiration is carried on without painful perceptions to the patient. For with the diminished oxygenation of the blood, the chemical changes become less active, and as the intensity of the respiratory changes is directly proportional to the vital energy of each organic element of the body, the supply therefore suffices for the demand. Such mutual adaptations lead to an adjustment that is perfect within *physiological* limits, provided only time is allowed them for the accomplishment of their end. Much error has therefore been propagated as to the influence of food and sleep upon asthma. The *normal* modification which these produce in the respiratory function has been constantly supposed to be an efficient provocative of dyspnœa. But the facts that seem to warrant this assumption have been falsely interpreted. The influence of food and diet in general, will be subsequently explained. For the present it suffices to say that sleep, as such, is by no means responsible for the nocturnal appearance of the dyspnœa. Floyer imagined that his asthma was produced in that way, and many gratuitous theories have since then been advanced in support of his view as to the occurrence of the paroxysms at night. A causal connection between

these events is so generally accepted that the fact of a dyspnœa rousing the patient from his sleep is supposed to be pathognomonic of a nervous disturbance. Hyde Salter thinks that sleep produces insensibility to respiratory arrears and that it exalts reflex action.[1] Physiology affords no evidence in favour of this theory. What is known of the subject is that, during sleep, respiration is retarded, and the absolute quantity of carbonic acid exhaled is diminished by one-fourth or even more.[2] But these phenomena indicate merely a temporary abatement of the vital energy in order that its activity may be increased through the day by the consumption of the oxygen that is stored up during the night.

Those compensatory forces, however, have their limits. They prove inadequate in the face of an additional obstacle to respiration. In the healthy an impediment to the interchange of gases is readily overcome by means of the reserve fund, so to speak, at their disposal; but in the asthmatic that fund is exhausted by its constant employment, even under physiological conditions, to maintain the balance between expenditure and supply. Hence, under such circumstances, a slight cause produces a disproportionately great effect.

Thus, the accession of an obstacle, however trifling

[1] *Op. cit.*, p. 198.

[2] Scharling, quoted by Vierordt.—*Grundriss d. Physiol. d. Menschen.* 3 Aufl. Tübingen, 1864, p. 552.

in itself, is sufficient to provoke an asthmatic seizure. This, like every other form of dyspnœa, is primarily due to a disturbance of the interchange of gases. The want of oxygen and the accumulation of carbonic acid excite the centre of respiration, and from this stimuli are transmitted to the respiratory muscles. The pneumogastric nerves lessen the resistance which the "noeud vital" opposes to that irritation, and they distribute the activity of the muscles in such a manner as to conduce to an efficient ventilation of the lungs. Section of the vagus, although profoundly modifying the respiratory mechanism, has yet no influence whatever upon the amount of work which in a given time is performed by the centre of respiration.[1] Nor has irritation of that nerve any effect upon the medulla oblongata, unless this be prepared for it by the gaseous contents of the blood.[2]

The dyspnœa peculiar to asthma is distinguished by its type. Expiration is relatively more difficult than inspiration. The reason is obvious. Inspiration is the result of muscular activity, so that the lungs yield to the traction which is greater than the resistance they are capable of offering. Expiration, on the contrary, is actively and mainly performed by the lungs themselves, and the want of their cooperation in this act cannot be fully compensated for by the aid of the auxiliary forces. Now, the predis-

[1] Rosenthal, *Athembusegungen*, etc., p. 75-125.

[2] *Ibid.*, p. 256.

position to asthma consists of nutritive changes of the smaller tubes and of the air-vesicles. These portions of the organ are truly the *loci minimæ resistentiæ;* they are the parts first and chiefly affected by noxious agents, and are thus readily disabled in the performance of their function. The intensity of the subjective symptoms is in a great measure attributable to the same cause. Even in the healthy, an obstacle to expiration produces *far greater* distress than one to inspiration, or to both phases.[1] But here, the lungs are, on account of their diminished resistance, widely distended, and this distension considerably increases the difficulty of expiration.[2]

The immediate causes of asthma are numerous and of various kinds, but all productive of the same results —obstruction of the bronchi. They are exhibited in the following table :—

I. Hyperæmia and œdema of the lungs.
   - *a.* Relaxative { Inhalation of irritating gases; Inhalation of dust; Thermal influence.
   - *b.* Collateral { Meteorismus; Embolism of the pulmonary artery.
   - *c.* Reflex paralytic.

II. Bronchitis.

III. Stenosis of the bronchi { Foreign bodies. Compression.

[1] Paul Bert, *op. cit.*, p. 413.

[2] Horvarth, "Zur Lehre von der Elasticität."—*Centralblatt f. d. Mediz. Wissenschaft.*, No. 48, 1873.

1. *Hyperæmia and Œdema.* The dilated capillaries, and more especially the vascular papillæ, which, as above described, project from the surface of the mucous membrane, are for various reasons greatly susceptible to fluxionary hyperæmia. Such turgescence readily causes occlusion of the bronchioles. The inspiratory traction, however, is soon increased in force, so as to enable it to effect the entrance of air into the lungs. The dyspnœa that nevertheless accompanies the hyperæmia is, therefore, due not so much to the absence of air as to the rapidity with which the blood passes through the dilated capillaries. There is no time available for the interchange of gases, and the blood thus arrives at the pulmonary veins in almost the same condition as it left the pulmonary artery. A similar process takes place in the parotid, in which, after section of the sympathetic, the blood in the veins presents the arterial character. The arterial system is thus supplied with blood imperfectly oxygenated. Meanwhile, air continues to enter the lungs, whereas none, or very little, can escape from them. The thorax is gradually distended to its utmost limits and is held in the extreme inspiratory position by the tetanic contractions of the respiratory muscles; the stimulus to these contractions being furnished by the carbonic acid in the blood. When the dyspnœa has reached its climax, amelioration generally takes place. It appears that the carbonic acid in the blood causes contraction

of the pulmonary capillaries, either by irritation of the vaso-motor nerves or of the vascular walls.[1] The current of the blood once being slackened there is now no obstacle to the ventilation of the lungs.

This form of hyperæmia is generally of short duration and does not give rise to exudation. But when the left ventricle is incapable of propelling all the blood that by the increased afflux is brought to it, a passive congestion complicates the fluxion. In consequence of the greater pressure, serum now readily transudes into all the interstices of the lungs. Such œdema most frequently occurs in hydræmia, especially of parenchymatous nephritis, and arises also from the temporary increase of the volume of the blood by an excessive accumulation of fluid in the system.[2]

a. Relaxative. Fluxionary hyperæmia is caused by irritants, which act directly upon the muscles or the ganglionic apparatus of the blood-vessels and causes them to dilate. It arises from

1. *The inhalation of irritating gases.* Some asthmatics believe that the air of certain places is particularly obnoxious to them, and they attribute this injuriousness to subtle atmospheric conditions. There is no doubt that every open or confined space, every field, and every house in town and country, possess what may be called their own peculiar climate. But

nothing justifies the assumption that the *normal* variations of the atmosphere are capable of producing dyspnœa, however delicate the bronchial surface may be. The mystery that surrounds these cases can, however, be readily solved; for when the "inscrutable differences of air" are responsible for the asthmatic seizures, their hurtful influence proceeds from the presence of irritating gases. Hyde Salter reports,[1] "Mr. C. has been asthmatic from his youth, and suffered at times severely; but for the last four years, during which he has lived in the same house, has had no attack. Last week he removed his residence about four or five hundred yards, to a higher spot, but more closely built round, and *very near to the exit of a sewer;* the third night he slept in his new abode he had an attack of asthma, and is suffering from a repeated attack at this moment." The exciting cause of this case is so evident as to need no mention; yet the conclusion that is drawn from it is that "the change of locality here was only a distance of four or five hundred yards, but it was sufficient to light up the disease that had been in abeyance for years."[2]

A temporary exposure to noxious gases, which, from decaying organic substances, or in the process of the various manufactures, are discharged into the open air, hardly produces an ill effect, because the space is so vast and the movement in it so continuous that those vapours are rapidly diluted, and thus rendered harmless. Dys-

[1] *Op. cit.*, p. 292. [2] *Op. cit.*, p. 293.

pnœa, however, results when their ascent is impeded so that a quantity of them remains within the zone from which the inspiratory current is immediately drawn. In this way fog exerts its injurious influence on the diseased mucous membrane of the respiratory passages; for, besides ill defined hydrocarbons, it contains large quantities of ammonia, of which Boussingault once found the enormous proportion of one grain in thirty-five ounces of the condensed water.[1] Considering that the injection of a few drops of ammonia into the bronchi causes the most intense pneumonia, the irritating effect produced by fog is readily intelligible.

The air of bed-rooms, especially in the winter, proves, perhaps, the most frequent cause of the "nocturnal" asthma. Notwithstanding the diminished energy of all the vital changes during sleep, at least one thousand cubic feet of air pass every hour[2] through the lungs, and return from them charged with more than four per cent. of carbonic acid, and completely saturated with water vapour; and the expired air contains ammonia, probably from decayed teeth, or from particles of food decomposing in the mouth, also hydrogen, hydrocarbons, and sulphuretted hydrogen, which in consequence of a faulty digestion may diffuse themselves into the intestinal veins, and be eliminated

[1] Rob. Angus Smith, *op. cit.*, p. 236.

[2] Krieger, "Ueb. Entstehung d. entzündl. u. fieberhaften Krankheiten".—*Zeitsch. f. Biologie*, 1869.

by the breath.[1] The products of perspiration and those derived from the combustion of candles, lamps, or gas, contribute in their turn to increase the insalubrity of the place. It is known that the air which contains one per mille of carbonic acid is irrespirable, and its injuriousness is due, not so much perhaps to that gas itself—which when pure, may without harm indeed be inhaled in a somewhat larger quantity than is then present—but to the organic substances which always accompany it. Yet even a slight excess of the pure carbonic acid causes turgescence, a sensation of heat and pricking in the conjunctival and respiratory mucous membranes, while at the same time it increases the cutaneous and mucous secretions.[2] In the presence of organic substances, however, a much smaller quantity of it becomes highly irritative. The inflamed mucous membrane of the respiratory tract seems particularly susceptible to its influence, and Hauke is led to conclude that the coughing fits of pertussis are traceable to that source.[3] The manner in which the organic substances act is as yet obscure. If it were permissible to speculate on the subject, the dyspnœa, which they undoubtedly produce, may perhaps be attributed to the "oxysulphide of carbon", which, according to

[1] Joh. Ranke, *op. cit.*, pp. 475, 482.

[2] Hermann Eulenberg, *op. cit.*, p. 67.

[3] "Inhalations Versuche mit verschiedenen Gasarten bei Tussis convulsiva."—*Jahrb. f. Kinderheilk.*, vol. v, Heft 1, 1862.

Radziewsky,[1] arises from putrifying organic substances, and is widely spread in nature. Radziewsky himself, on inhaling oxysulphide of carbon, immediately felt a pressure on the head, a tendency to vertigo, and constriction across the chest; there was, moreover, the sensation as if the air-passages were obstructed. He soon, however, recovered on exposure to the open air. Experiments on animals gave the same results—dyspnœa and great distension of the thorax. Now, however large a bed-room may be, a few hours' occupation of it will render it insalubrious in the manner just mentioned, unless the air be constantly renewed to the extent of at least two thousand cubic feet per hour and person. How imperfect even such ventilation must be, as regards the removal of the gaseous impurities, in comparison to their dilution out of doors, is evident from the fact that here more than six millions cubic feet of air are available for the purpose.[2] Unfortunately asthmatics, as a rule, adopt no measures for the supply of fresh air. They rely on natural ventilation, or persuade themselves, if this matter even receives their attention, that opening the door of the bed-room is all that is wanted. But the consequence of that neglect is that on account of the slow diffusion of gases[3] the patients are sur-

[1] "Die giftigen Wirkungen d. Kohlenoxysulfids."—Virchow's *Archiv*, Bd. 53, 1871, p. 370 *et seq.*

[2] Joh. Ranke, *loc. cit.* [3] Rob. Ang. Smith, *op. cit.*, p. 536.

rounded by the noxious exhalations which they themselves produce. As the temperature of the room is raised by respiration, foul gases from kitchen sewers and other sources are thus attracted into the room more readily than fresh air. After several hours, the atmosphere becomes so vitiated, that respiration is impossible ; so that towards morning the patient is roused from his sleep by an attack of asthma, and he is forced to seek relief at the open window.

2. *Inhalation of Dust.* Foreign bodies, of different kinds and in various quantities, are at all times present in the atmosphere. The dust in the open air is composed chiefly of inorganic particles, and amounts, as for instance in the streets of Paris[1], to about one-sixth of a grain in every cubic foot. Indoors it mainly consists of organic substances and more especially of living organisms, whose development and multiplication here meet with favourable conditions. So long as those solid particles are undisturbed and are permitted to follow their own gravitation their tendency is to settle on some object, and thus far they are harmless. But when an external moving force returns them into the air, they may be brought within reach of the inspiratory current, and thus made to enter the air passages. Their effects vary according

[1] Tissandier, quoted by Eduard Lichtenstein, "Ein Stückchen öffentlicher Gesundheitspflege, etc."—*Berl. Klin. Wochensch.*, No. 46. 1874.

to their nature and quantity. Fragments of coal, of iron, of stone, and of glass lacerate the mucous membrane; hairs, feathers, fibres of wool and of cotton, once impregnated with moisture, firmly adhere to the secreting surface, and produce great irritation. Infusoria, of which fifty-six species have been observed in the air, are raised by the wind from drying waters and carried great distances; and though apparently dead, they begin, in contact with the mucus, to loosen the covers which enclose their withering bodies and probably regain a new vitality.[1] Algoid cells and fungi are particularly abundant in ill-ventilated rooms; living, and ready to undergo development on meeting with suitable conditions, they rapidly germinate, or are already germinating, when they are deposited in the air-passages;[2] some of them form a mycelium or masses of toruloid cells; others produce their characteristic heads of fructification. One circumstance connected with the life of fungi deserves here, perhaps, a passing notice. On watching Botrytis cinerea, taken from a rotten apple, Rindfleisch found that the formation of its spores took place only at night. Subsequent observation has fully confirmed this exclusively nocturnal growth. The

[1] Humboldt, *Ansicht d. Natur;* quoted by Ehrenberg. *Abhdl. d. Königl. Akad. d. Wissensch.* Berlin, 1872, p. 233, *et seq.*

[2] Cunningham, "Microscopic Examination of Air."—*Ninth Annual Report of the Sanitary Commissioners.* Calcutta, 1872.

want of light had not the same effect, for the spores kept in darkness did not proliferate.[1] But whether the organic substances that accumulate during the night in confined places, have any influence upon that phenomenon, must for the present remain a matter of conjecture : much less is there ground for supposing that the nocturnal appearance of the dyspnœa is connected with the greater vitality of these fungi at the time.

However, the result of such inhalation is invariably the same—fluxionary hyperæmia, and even inflammation, of the affected portion of the mucous membrane. The *colds* which *draughts* produce arise in that way. It is the current of air that lifts the particles of dust from carpets and curtains, from walls and furniture, and brings them in contact with the lining of the respiratory tract. The serious affections of the lungs which develope themselves from a prolonged exposure to foreign bodies in the air, are exemplified by the diseases to which knife grinders, masons, weavers, and other operatives are especially subject. Little regard, however, is paid to the "invisible nastiness", as Professor Tyndall[2] calls it, which constantly exists in ill ventilated rooms. The bronchitis so often attributed to "cold" is, in reality, due to the inhalation of frag-

[1] "Ueber d. niederen Organismen."—Virchow's *Archiv*, vol. liv. 1872.

[2] "Dust and Disease."—*British Medical Journal*. 1870, p. 118 *et seq.*

ments of glass and stone,[1] which, on post-mortem examination are found in the air-passages, or to the fibres of wool which are seen in the larynx and appear in the sputa.[2] Organic substances are even more powerful. The influence of Chætomium elatum and Penicillium glaucum has already been mentioned.[3] The accidental inhalation of a small quantity of flax meal has been known to produce intense bronchitis and dyspnœa.[4] The dust-like spores of a large puff-ball caused in the same way so great irritation of the air-passages that the patient was confined to his room for several days.[5] In isolated instances the appearance of an asthmatic seizure may be directly traced to foreign bodies in the air, such, for instance, as mustard, and ipecacuanha powder. In the majority of cases, however, the exciting cause escapes detection. But it is well to remember that asthmatics are particularly susceptible to that kind of irritation. Not only is

[1] F. Pouchet, "Recherches sur les corps introduits par l'air dans les organs respiratoires des animaux."—*Compt. Rend.* 1860, tom. l, p. 1121 *et seq.*

[2] Sommerbrodt, "Nachweis einer neuen Ursuche z. Erkrankungen d. Athmungsorgane."—*Berlin. Kl. Wochensch.*, No. 7, 1870.

[3] Blackley, *op. cit.*, p. 58.

[4] Henry Kennedy, "A Case in which a Disease like Measles arose from an Unusual Cause."—*Dublin Quarterly Journal of Medical Science*, vol. xxxviii. 1863.

[5] M. C. Cooke, *Fungi; their Nature, Influence, and Uses.* Edited by the Rev. M. D. Berkeley. London, 1875, p. 216.

their respiratory surface always more or less diseased, but the chronic congestion of the nasal mucous membrane, or the presence of a nasal polypus, forces them to breathe through the mouth, and thus deprives them of the important filtering apparatus. When the nose is pervious, foreign bodies do not reach further than the pharynx; but when that canal is obstructed, they readily enter, not only the bronchi, but are found also in the substance of the lungs.[1] Pulverulent bodies penetrate even to the bronchial glands.[2]

The inhalation of pollen gives rise to the peculiar symptoms known as *hay-fever* or hay-asthma. Ever since attention has been directed to them, their origin has been connected with the hay season, but has been erroneously attributed to the influence of heat and light, or to the odour of certain plants. Mr. Wyman persists in this error, and still maintains that hay-asthma is a purely nervous affection.[3] But the researches of Dr. Blackley[4] have conclusively established the dependence of the disease upon the presence of pollen in the air. He found that from the end of May to the end of July, the atmosphere of certain localities

[1] Fournié, "De la pénétration des corps pulvérulents, gazeux, volatiles, solides," etc.—*L'Union Médicale.* 1861, tome xi, pp. 582, 598.

[2] Knauff, "Das Pigment d. Respirationsorg."—Virchow's *Arch.* Vol. xxxix, 1867, p. 449 *et seq.*

[3] "Autumnal Catarrh."—*Boston Med. and Surg. Journ.*, August 19, p. 209, 1875.

[4] *Op. cit.*

contains pollen grains in varying quantities. A small portion of that substance, even less than one-two-hundredth of a grain in weight, causes, if directly applied to the mucous membrane of the nose, within five minutes, occlusion of the nasal passages with sneezing and lachrymation. The inhalation of a larger portion of it is invariably attended by a sharp attack of asthma. The severity of this abates with the diminished production of the agent, and disappears altogether when this ceases to be generated. This irritating effect is possessed by the pollen of all plants, but more especially by that of the Graminaceæ, or among the cereals, by that of rye.

The action of pollen upon the mucous membrane may perhaps be explained by its behaviour in contact with water-vapour, and also with mucus.[1] Under the microscope, pollen appears as a simple cell, with granular contents, and a cell-wall, consisting of an outer and an inner membrane. The extine is coated with an oleo-resin, of a rich amber, or, at times, a pale straw-colour, and contains several slits or pores, across which the intine is stretched so as to impede the escape of the granular matter. It does not appear that the oleo-resin has any specific action beyond enabling the grains to adhere to objects to which they may approach. On the addition of fluid, the cell is distended;

[1] Fr. Lühe, "Zum Heufieber."—*Deutsch. Arch. f. Klin. Mediz.* Vol. xiv, H. III and IV, p. 426 *et seq.*

the inner membrane projects through the slits of the outer in the shape of small mastoid processes, to which the granules are attracted. A further distension causes the membrane to burst, and the granular contents are, with a considerable force, ejaculated into the surrounding fluid. These granules irritate the mucous membrane, especially the mucous glands, and reappear finally in the sputa, resembling zoogloœa masses.[1]

Dr. Salisbury[2] attributes the cause of hay-asthma to the presence of an animalcular organism, the *asthmatos ciliaris*. This parasite is said to inhabit stagnant as well as running waters, and to develope itself also on fermenting organic matter. It is said to be spherical or oval in shape, and to consist of a simple sac, with one or more large nuclei, and many smaller granules of various sizes. The sac itself is armed with cilia, and, between them, with a long proboscis. The young are developed within the parent cell, and are discharged from the end opposite the cilia. Dr. Salisbury's statements, however, have not yet been confirmed by any other observer.

E. Lewy[3] observed asthmatic seizures as an isolated affection in 26 out of 1,186 cases of saturnismus. He attributes the dyspnœa to the lead dust, which, from

[1] Lühe, *loc. cit.*

[2] "Infusorial Catarrh and Asthma."—Hallier's *Zeitschrift für Parasitenkunde*, vol. iv, Heft 1, 1873.

[3] "Seltene Formen d. Bleivergiftung."—*Oest. Zeitsch. f. prak. Heilk.*, vol. vi, p. 27, 1870.

the packing of white lead and the cleaning of type cases, escapes into the air. The inhalation of the lead dust is immediately followed by dyspnœa, palpitation, stitches in the diaphragmatic region, and cough. In from six to seven hours the typical attack of asthma is developed. Lead appears to contract the vessels upon which it acts, while those that escape its influence become congested.

3. *Thermal influence.*—There is a widespread belief that "cold", by inducing bronchitis, is a frequent provocative of asthma. Every patient knows instances, where he himself or his friends suffered from dyspnœa, which he could account for only by a loss of temperature. His opinion fully accords with the prevalent doctrine on that point. Clinical observers of the highest repute constantly advert to the danger of a chill, and maintain that, even in well appointed hospitals, a patient in bed may "catch cold" (Lebert). Indeed, so powerful and so ubiquitous is this agent, that, according to the text books of medicine, it is at the root of almost all diseases, from cerebro-spinal meningitis to diarrhœa ; from acute rheumatism and endocarditis to a sneezing fit.

Formerly it was supposed that a chill checked the cutaneous perspiration, and that an excrementitious substance, retained in the blood, produced inflammation of internal organs. The varnishing of animals, and the serious symptoms that generally follow these

experiments, unless proper precautions are taken, were supposed to confirm that opinion.[1] No poisonous substance, however, was found in the blood, and it was soon admitted that the symptoms of varnishing do not correspond to those of "colds". That theory therefore was abandoned. At present the cause of the disease is sought in disturbances of the economy of heat. Two different views are held on the subject. According to the one, a *sudden* change of temperature, however slight it may be, produces one-sided radiation of heat, and leads through the vaso-motor nerves of the skin to circulatory derangements of internal organs. Professor Pettenkofer insists on the *suddenness* with which the loss of temperature is effected. The difference between the rapid and the gradual abstraction of heat may, in his opinion, be compared, so far as the results are concerned, to a man going or falling down stairs.[2] But if one-sided radiation had really such injurious consequences, there could not be a more dangerous instrument than a fan in the hand of a lady. As a matter of fact, it is not so dangerous as alleged; and the reason is that if, adopting the simile, the man were of caoutchouc, or, at any rate, provided with the same elasticity as the regulators of temperature are endowed with, it would be perfectly immaterial, so far as the conse-

[1] Edenhuizen, *Zeitsch. f. rat. Mediz.*, C. xvii, p. 35.

[2] *Beziehungen d. Luft. z. Kleidung., Wohnung und Boden.* Braunschweig, 1872, p. 73 *et seq.*

quences are concerned, in what manner he descends. Rosenthal[1] equally regards the *suddenness* of the changes as the cause of "cold". His experiments led him to conclude that when an over-heated animal, in which the cutaneous capillaries are paralysed, is suddenly exposed to a lower temperature, the blood is rapidly cooled, and in that condition it is returned to the internal organs. It then produces inflammation of those parts that are "loci minoris resistentiæ". In man, a *rise* of temperature follows the application of cold to the heated surface,[2] and there is little doubt that the same law holds good also in animals. But the *sudden return* of the blood implies a contraction of the cutaneous vessels; and, as their paralysis is essential to the cooling, there cannot be a great loss of heat; or, if the vessels were paralysed to the extent that they have lost their physical property of contracting upon the application of cold, no blood could be returned from the surface to the centre. Without entering upon the questions that are involved in this subject, it suffices to say that over-heated (*i.e.*, febrile) bodies *do not catch cold.* A reduction of their temperature tends to re-establish the healthy balance. The success of modern anti-febrile treatment is based upon

[1] *Zur Kenntniss d. Wärmeregulirung.* Erlangen, 1872.

[2] Liebermeister, *Ueber Wärmeregulirung u. Fieber;* Volkmann's *Samml.*, No. 19, p. 121; Garrod, *Journ. of Anat. and Physiol.*, 1872, No. ix; Schmidt's *Jahrb.*, clxiii, p. 93.

that principle, which a thousandfold experience has shown to be correct.

According to another view, "colds" arise from an appreciable loss of temperature, such as is produced by currents of air and copious evaporation from the perspiring skin.[1] Hirsch concludes that "colds" are most frequent when the temperature and the dew-point of the air approximately coincide, as in spring and autumn.[2] The opinion of Hirsch is, at any rate, based on very extensive statistics; and it is deduced from incongruous facts, which allow no other generalisation. Yet meteorological reports, on however vast a scale, are alone insufficient to solve this obscure problem. At the present stage of civilisation, every individual creates for himself, or otherwise obtains, an atmosphere in which he thrives or decays. The state of the weather will undoubtedly affect, in a different manner, the inhabitant of Belgravia and of Bethnal Green. Both may bestow the same anxious and efficient care to ward off the cold; yet, while the one escapes unhurt, the other catches bronchitis. The influence of the weather would here be very remote. Its injuriousness would be admissible only if it could be shown that those who most expose themselves to

[1] Friedrich Falk, *Ueber Entstehung von Erkältungs-Krankheiten;* Reichert u. Du Bois-Reymond's *Arch.*, 1874, p. 159 *et seq.*

[2] *Handb. d. histor.-geograph. Pathologie.* Erlangen, 1862-1864, Bd. ii, p. 2-19.

the supposed cause, are also the most frequent sufferers of its assumed consequences; and herein statistics not only fail, but prove just the opposite.

These considerations are dictated by what daily experience suggests, and they are decidedly adverse to the prevalent doctrine. It is needless to refer to those well known instances where delicate as well as strong persons daily sustain, deliberately or unconsciously, considerable losses of temperature. Yet in no single instance has such loss *alone* produced inflammation of an internal organ. The question is not whether cold can, under no conditions, be hurtful. Nobody doubts that, if an animal be kept in a tin box surrounded with ice, the temperature of the body will decrease to 18 or 20° C., and that death may thus ensue from paralysis of the heart and œdema of the lungs.[1] These are *experiments* that happily have their counterpart but rarely in practice. But that is not the point at issue. The supposition is that if a few square inches of skin were denuded of their customary covering, and exposed even to the warm temperature of a room, the consequence would be an inflammatory affection of the muscles or of an internal organ.[2] Let the worst that may, happen in such a case. Let the blood in that circumscribed space, and even to a certain depth, exchange its temperature with that of the

[1] Walther, Virchow's *Arch.*, vol. xxv, p. 414 *et seq.*

[2] Seitz, in Ziemssen's *Cyclopædia*, vol. xvi, p. 232, *et seq.*

atmosphere. Let the vessels at that spot be contracted, and the mass of blood in other organs be correspondingly increased. What can follow from all this? The body produces more heat than it can retain, so that the loss is readily compensated. The increase of the vascular pressure would not sensibly influence even that of the muscles of the extremities, where such differences are generally balanced; and since ligation of the abdominal aorta does not produce hyperæmia of the lungs, it can hardly be expected that the quantity of blood displaced from a few square inches of skin could have that effect. Granted even that a hyperæmia resulted, there is still a wide difference between the temporary turgescence and inflammation. But facts are altogether opposed to that theory. Jürgenssen rapidly reduced the rectal temperature of a man to 33° C.; but this man, neither at the time nor subsequently, felt any inconvenience from the experiment to which he submitted himself.[1] Another, having spent a whole winter's night in the open air at a temperature of—6° C., is brought to the hospital with a rectal temperature of 24° C.; and within thirty-six hours he leaves the institution in perfect health (Nicolaysen). Conceding that the favourable issue of both these cases may in a great measure be attributed to individual peculiarities, still it shows not the less that the fatality generally

[1] *Die Körperwärme d. gesunden Menschen.* Leipzig, 1873, page 30.

ascribed to cold is greatly exaggerated. There is, of course, the unanimous assertion of almost all patients that they can distinctly trace their disease to the influence of that agent. But much of this opinion is, doubtless, due to the circumstance that the laity and physicians are accustomed to consider the presence of a bronchial affection, more especially, as a cogent proof that the patient *must have caught a cold.* And under these circumstances it is not difficult to recall the occasion when the cooling has taken place. Much, however, is attributable to this, that all inflammatory affections, whether slight or intense, local or constitutional, are ushered in, or are often for days preceded, by chilliness or rigors, while the causes of these (*i.e.*, the local changes) only subsequently appear. No disease is attended by more pronounced constitutional symptoms than pharyngitis; and here the febrile movement, cold followed by heat, is felt for days before the difficulty in swallowing is noticed. Thus the first expression of the disease is often taken for its cause; and this mistake will be readily intelligible from the disagreeable sensation which cold in general occasions.

Is then the economy of heat really disturbed so easily as supposed? Are the regulators of the temperature so powerless towards even a slight disturbance? Surely the regard to the compensatory forces—the operation of which no organic processes exhibit in so striking a manner as those connected with animal

heat,—suggests a negative reply. Amidst the most varying external and bodily conditions, the healthy organism preserves its own daily temperature at an average height of 37.2° Cent. Within wide limits, every loss of heat is compensated for by an increased production, and every excess of heat is rectified by an increased expenditure. This regulation proceeds from an excito-caloric and a moderating centre, to which thermal stimuli are probably conveyed from the periphery.[1] But it is carried out by the cutaneous capillaries, and here it is subject to the physical laws of temperature : cold contracts the vessels of the skin, and forces the blood to the centre, or rather into the muscles ; heat, on the contrary, dilates the vessels and favours perspiration.

It depends therefore upon the energy of the vital, functions and the persistence and intensity of the disturbing element, how far the limits of the compensation reach. It is evident that those limits must necesssarily vary in different individuals and at different times. Upon the whole, the body is better able to withstand cold than to dispose of its superabundant heat. The living organism produces, irrespective of food, two

[1] Röhrig u. Zuntz, "Zur Theorie d. Wärmeregulation."—*Arch. f. d. ges. Physiol.*, Bd. iv, 1871, p. 57 *et seq.* Riegel, "Ub. d. Einfluss d. Centralnervensystems auf die thierische Wärme."—*Arch. f. d. ges. Physiol.*, vol. v, 1872, p. 629-670.

million calories in twenty-four hours; and this supply may be maintained or increased by various means, so as to meet every conceivable emergency. The difficulty at times is, as already mentioned, to remove the surplus, which, if accumulated in the body, would, in twenty-four hours, raise the temperature to the fatal degree of 48° C.[1] The efforts in one direction are invigorating; those in the other have a debilitating effect. The local action of heat and cold presents the same difference, inasmuch as a slight increase of the temperature is destructive of the tissues, while even a considerable reduction of it merely renders their vitality latent. The frozen muscles of the frog retain, when thawed, their electrical irritability (Kühne). A comparatively slight increment of heat, on the contrary, coagulates the myosine of muscles and destroys their vital properties. Similar changes are observed in the blood; extreme degrees of cold are to the life of the red blood-corpuscles, far less prejudicial than is even the febrile heat of the body.[2] A rabbit's ear may, with perfect impunity,[3] be exposed to a temperature of—6° C. for from twenty-five to thirty minutes. Its exposure to a temperature of from + 46 to + 49° C. is attended by œdema of the surrounding tissues, and at + 50° C. the emigration of white

[1] Liebermeister, *loc. cit.*

[2] Manassein, *loc. cit.*, pp. 25 and 28.

[3] Cohnheim, *Neue Untersuchungen über d. Entzündung.* Berlin, 1873, p. 54.

blood-corpuscles commences.[1] The reaction of a rabbit's ear naturally differs from that of a human lung, and the changes observed in the former allow no such direct inference as those that may occur in the latter. Still these experiments justify the conclusion that the "cold" to which the lungs are exposed cannot possibly have the noxious consequences usually attributed to it.

Although the injurious influence of cold has been grossly exaggerated, yet it cannot be denied that asthmatics may suffer from dyspnœa, in consequence of a considerable loss of their temperature. Their organs of respiration and circulation would not only be greatly taxed, but might even fail to meet the consequent increased demand for oxygen.[2] In what manner the pulmonary surface is, under such circumstances, affected, is not quite known. The face and hands of delicate individuals exposed to cold become cyanotic, because the arterioles are contracted, and the veins are gorged. But it is impossible that in the lungs the arteries can be so contracted or the veins so gorged, as to produce the same degree of cyanosis. For, not only is the inspired air warmed on its passage through

[1] Cohnheim, *ibid.*, p. 54.

[2] Leichtenstern, "Ueber das Volumen d. unter verschiedenen Umständen ausgeathmeten Luft."—*Zeitsch. f. Biolog.*, 1871, Bd. vii, p. 179 *et seq.* Speck, "Untersuch. über Sauerstoffverbrauch", etc.—Virchow's *Jahresbericht*, 1871, vol. i, p. 67 *et seq.*

the nose, but the bronchial surface is far more vascular than the skin, and much nearer to the centres of the production of heat.[1]

Over-heating is a more potent and also a more frequent cause of dyspnœa than cold. It is well known that asthmatics greatly suffer in hot and sultry weather. Trousseau considers asthma as a disease most prevalent in hot climates and in the summer;[2] but his observations are not quite to the point, inasmuch as they include also *hay* asthma, which he ascribes to heat and light, instead of the inhalation of pollen. The dyspnœa must be attributed to the imperfect disposal of the superabundant heat when the warm atmosphere is saturated with moisture. The pulmonary surface is probably then in a similar œdematous condition as the skin. The heated blood irritates the centre of respiration,[3] and also that of the vagus (Cyon). The volume of the expired air increases, but the exhalation of carbonic acid diminishes, not on account of its deficient formation, but because the oxygen loses, in a ratio corresponding with the rise of temperature from 0° to 37° C., the power of expelling that gas.[4]

[1] Albert u. Stricker, "Untersuch. über d. Wärmeökonomie d. Herzens u. d. Lungen".—*Wiener Mediz. Jahrbuchen*, 29-50.

[2] *Op. cit.*, vol. ii, p. 452.

[3] L. Goldstein, *Ueber Wärmedyspnœ. Verhandl. d. physik-mediz. Gesellsch. zu Wurzburg.* Neue Folge, Bd. ii, 1872, pp. 156-169.

[4] Donders, "Le chimisme de la Respiration", etc.—*Archives Neerland*, vii, p. 193. Virchow's *Jahresbericht*, 1873, p. 161.

Over-heating is not a less active cause of asthma in the winter season. There are numerous persons, in all stations of life, who at all times consume more than the necessary quantity of food, who, particularly in the winter, over-burden themselves with warm clothing, and spend their time in hot and ill-ventilated rooms at the fireside. That food, and *à fortiori* an excess of it, as well as the products of the faulty digestion, do increase the bodily temperature, has been abundantly ascertained by exact thermometric measurements. Besides, the flushing of the face, the throbbing of the arteries, and the palpitation of the heart, from which those individuals generally suffer after meals, sufficiently indicate a febrile movement. Yet the disposal of their superabundant heat is a matter of considerable difficulty. The loss from the lungs is slight, because the air of the rooms is thoroughly warmed. The skin, even if no thick layer of subcutaneous fat impedes radiation[1]— and the obese are the most numerous among that class of persons—is, on account of the great amount of clothing, surrounded by an atmosphere of almost the same temperature as that of the body. Among those who seek advice at hospitals, it is often seen that even the robust men are in the habit of wearing in the winter— 1, a thick overcoat; 2, a frock coat; 3, a thick waistcoat; 4, a flannel shirt; 5, a flannel vest; and 6, a chest pro-

[1] F. Kluge, "Untersuchungen über d. Wärmeleitung der Haut". —*Zeitschrift f. Biolog.*, Bd. x, p. 73.

tector or "cat's skin", all these "to keep their chest warm". And the same anxious care is bestowed upon the clothing indoors. Under these circumstances nature relieves itself by copious perspiration, and thus, by the evaporation of the perspired water, endeavours to reduce the temperature. The disagreeable sensations that are apt to arise from this cooling process, are regarded by the patient as a sure sign that "he must have somehow caught a fresh cold". And such is the vicious circle, that the means which he now adopts to combat, only increase the evil. But although the regulators of the temperature succeed in preserving it for a length of time at its normal height, there is little doubt that they ultimately succumb to the prolonged strain upon their energy. This failure will not appear unexpected, if it be remembered that the main spring of that regulation are the cutaneous arterioles, or rather their muscular coats, whose continued relaxation would, even in the absence of any other cause, greatly derange their nutrition. If this much be admitted, it is needless to trace the steps that lead to the retention of heat in the body—a truly sub-febrile condition. Now, it is known that in insolation, death is due to the degeneration of all parenchymatous organs, and especially of the heart.[1] The danger in acute infectious diseases arises from analogous causes. Experiments on animals,

[1] Obernier, *Der Hitzschlag.*, 1857, quoted by Wagner; *Allgem. Pathologie*. 1868. p. 84.

by exposing them to a high temperature for some time (37° C. for thirty-six hours),[1] are attended by the same results. Not only the liver, the spleen, and the kidneys, but the entire muscular system, including the coats of the arterioles, undergo fatty or similar degeneration, these changes being then particularly marked in the substance of the heart.[2] As such derangements of nutrition are invariably produced by a *high temperature* of only a *short duration*, it cannot be doubtful that the prolonged action of a *lesser* degree of heat must have similar consequences. Indeed, it appears that such is the case in those who most emphatically declare their susceptibility to "catching cold". One acquainted with their habits of life, will readily concede that they seldom or never give *cold* the least chance of exerting its injurious influence, but that they suffer from the consequences of overheating. Their vessels are dilated and degenerated; serum, and even white blood-corpuscles escape on the slightest provocation. A heated room, the irritating gases—the products of respiration and combustion, such as accumulate in the air of a theatre—and the dust which a current of air, *the draught*, raises from the ground:

[1] Litten, "Ueber d. Einwirkung erhöhter Temperaturen auf den Organismus."—Virchow's *Arch.*, vol. lxx, 1877, p. 12.

[2] Iwashkewitsch, "Ueber d. pathol.-anatom. Veränd. d. parenchym. Organe unt. d. Einfl. hoher Temperat."—Virchow's *Jahresb.*, 1870, vol. i, p. 179.

these suffice to produce the supposed effects of "*catching cold*".

b. Collateral.—As the collateral hyperæmia is always the necessary consequence of an ischæmia of the lungs, respiration is thus at once impeded, both by the diminution of the blood in one place, and its corresponding accumulation in another. Intense dyspnœa may, therefore, be produced by—

1. *Meteorismus.*—Asthmatics suffer, as a rule, from the severer forms of dyspepsia. The venous congestion which, as previously pointed out, spreads in the course of time also to the intestinal mucous membrane, in consequence of the impeded circulation through the lungs, is the fruitful soil of catarrhal inflammation, so that derangement of digestion becomes as constant a symptom of asthma as the dyspnœa itself. If the quantity of the food is too large, or if its quality is unsuitable, the abnormal fermentation which now takes place, gives rise to a copious generation of carbonic acid and of hydrogen, besides other gases. In this way, the intestines may be so greatly distended, that the diaphragm is pushed upwards to the fourth and even third ribs, and compresses in that position the bases of the lungs. Ancient and modern literature contain the record of cases in which extreme flatulence proved even the immediate cause of death.[1]

[1] Bamberger, "Krankh. d. chylo-poietisch. System."—Virchow's *Handb.*, Bd. vi, Abth. 1, 1864, p. 212.

Some articles of food, as sugar and those containing saccharine matter, and fermented liquors, are particularly obnoxious, on account of the readiness with which they are decomposed into carbonic acid and hydrogen in contact with putrifying albuminous substances. Old and decaying cheese has, more or less, the same effect. It appears that the fungi which it contains, harmless though they are to a healthy digestion, excite fermentation when the gastric juice has lost its antiseptic properties.

Constipation is an equally frequent cause of meteorismus. Even in the healthy, there is, according to Fabius,[1] a considerable difference in the volume of the expired air before and after defæcation—a difference that would, for obvious reasons, be still greater in the asthmatic.

2. *Embolism of the Pulmonary Artery.*—In the feeble, and cachectic asthmatics of all ages, circumstances are unusually favourable for the formation of thrombi. In the dilated veins most distant from the centre, as the femoral and saphenous, and in those also of the abdomen, as, for instance, the uterine plexus, the blood readily clots in consequence of its slackened current, and of the weak action of the heart. If a thrombus is once formed, portions of it may be detached and impacted in the pulmonary artery or its branches. A slight muscular effort, or an excitement

1 Wintrich, *loc. cit.* p. 100.

of the circulation, suffices for this purpose; but, occasionally, an embolic obstruction is produced without appreciable cause even during sleep.[1] The consequences vary according to the size of the occluded vessel.[2] If this is one of the subordinate branches, the circulation of the territory which it supplies is readily re-established, and respiration is not disturbed. But when the embolus enters, as it most frequently does, the second or third subdivisions of the pulmonary artery (Paget and Virchow), occluding a considerable space, it generally gives rise to intense dyspnœa.[3] Much depends, as regards the origin and persistence of the dyspnœa, upon whether the cardiac action is energetic enough to propel a sufficient quantity of blood through the adjoining capillaries, by which alone the circulation in the obstructed part is maintained.[4] At best, however, this is only a slow process. Meanwhile, the collateral hyperæmia of the pervious portions of the lungs is accompanied by œdema, since the causes that favoured the production of the clot, equally facilitate the passage of serum from the vessels under the increased pressure. Occasionally,

[1] Gerhardt, "Der hæmorrhagische Infarkt".—Volkmann's *Sammlung*, No. 91.

[2] B. Cohn, *Klinik d. embolischen Gefässkrankheiten.* Berlin, 1860, p. 270, 273.

[3] *Conf.*, p. 61.

[4] Cohnheim und Litten, "Ueber die Folgen d. Embolie d. Lungenarterie."—Virchow's *Archiv*, Bd. liii, p. 99 *et seq.*

the œdema is the consequence of a hydræmia, as in the case of parenchymatous nephritis. In this case, a thrombus extends from the hilus of the kidney into the inferior vena cava,[1] and not infrequently the embolism of the pulmonary artery is the first indication of the renal disease.

c. Reflex Paralytic.—Violent emotion is, by some writers, mentioned as a provocative of asthma. Most of the cases, however, related in support of this view, allow also of another construction as regards their causation.[2] It is a matter, therefore, for future observation, whether or not mental excitement possesses the alleged influence. Should the present supposition be confirmed, the dyspnœa can arise solely from a hyperæmia of the lungs. The flushing of the face from psychical irritation suggests the possibility that a similar process may take place in the bronchial arteries,[3] a paralysis of the pulmonary vessels being precluded by the absence of a tonus. Or the excitement of the cardiac action may lead to congestion of the lungs, in consequence of the diminished resistance of their vessels. But all this, it must be admitted, is at present very obscure.

II. *Bronchitis.*—The bronchitis of asthmatics is,

[1] Virchow, *Gesammelte Abhandlungen*, etc., p. 233.

[2] Germain Sée, *loc. cit.* ; *Conf.*, p. 81 *et seq.*

[3] Comp. Dieffenbach ; mentioned by Virchow.—*Handbuch*, etc., Bd. i, p. 145.

as a rule, merely an acute exacerbation of a similar chronic process, and this circumstance explains not only the facility and frequency of its occurrence, but also the absence of marked constitutional disturbance. Insidious in its approach, its sole manifestation is intense and suffocating dyspnœa, which often lasts, with more or less perfect intermissions, for days and even weeks, long after the vascular engorgement may be reasonably supposed to have completely subsided.

The origin and persistence of that dyspnœa have, on account of the seeming inadequacy of the attendant physical signs, always been the subject of much speculation. But the apparent obscurity on those points admits of a ready solution, if it be remembered that the impediment of the respiration arises, not so much from the inflammatory process as from the inflammatory product. There can be little doubt that the textural changes of the mucous membrane, peculiar to asthma, are not without their influence on its function. Even such differences as normally exist in the linings of the larger and the smaller air-tubes affect, in an appreciable manner, the chemical constitution of the secretion ;[1] for, as the number of the mucous glands decreases with the diameter of the bronchi,[2] their specific activity cannot altogether be replaced by that

[1] Andral and Ch. Robin, article "Crachats", in *Nouv. Dict. de Med. et de Chir.-prat.*, t. x. p. 70.

of the epithelium alone. Thus it is, that in the atrophic emphysema and bronchiectasis, the inflammation of the *anæmic* mucous membrane produces, even when its glandular apparatus is intact, only a scanty and relatively dry exudation. To this cause the "catarrhe sec" mainly owes, as is well known, its distressing symptoms, and it scarcely needs to be mentioned, that these symptoms must be even more aggravated in the case of asthma. In the hypertrophous form of the disease the exudation is, on the contrary, very abundant; for the vascularity of the mucous membrane supplies a large quantity of blood, the constituents of which may easily escape through the altered walls of the vessels. But, as here the mucous glands are either destroyed or otherwise incapacitated in their function, the exuded material therefore retains, in a great measure, its original character. The serum and white blood-corpuscles, instead of being converted into mucus, coagulate and obstruct the bronchi. Essentially, this form of inflammation is a fibrinous (croupous) bronchitis; and Leyden consequently holds that this is in all probability the anatomical basis of the asthmatic seizures.[1] Leyden founds his opinion on the nature of a peculiar kind of crystals, which, evidently derivatives of white blood-corpuscles,[2] may be fre-

[1] *Loc. cit.*, p. 329.

[2] E. Neumann, quoted by Leyden, *loc. cit.*, p. 326.—*Brondegeest Centralblatt*, 1871, No. 9. F. W. Zahn, "Untersuch. üb. Thrombose".—Virchow's *Archiv*, Bd. lxii, p. 81 *et seq.*

quently observed in the sputa of asthmatics. Fibrinous bronchitis would thus appear to be far more frequent than is generally supposed. Indeed, the current belief as to its rareness ill accords with the fact that the fibrinous is merely an exaggerated condition of the catarrhal inflammation. But there are grounds for suspecting that the presence of the affection may be, and frequently is, overlooked. Its diagnosis depends altogether on the characteristic sputa.[1] When arborescent casts are expectorated, it is difficult not to recognise the nature of the disease. Yet, in some cases, the coagula are rolled up into pellets of the size of a pea, of a bean, or of a hazel nut, and are covered with mucus or streaks of blood;[2] so that their structure is discernible only when they are examined in water. In others, again, the plastic exudation is, for a time, retained within the bronchi, and here it is converted into mucus or into a puriform detritus, as it is in croupous pneumonia,[3] or in the thrombus of a vessel. The exhibition of an emetic may thus unexpectedly reveal the presence of a bronchial cast.[4] If, however, the process is allowed to proceed, the sputa may be deprived of their diagnostic

[1] Biermer.—Virchow's *Handbuch*, etc., Bd. v.

[2] Lebert, *Klinik. d. Brustkrankh.*, Bd. i, p. 119.

[3] Rindfleisch, *Lehrbuch*, etc., 1871, p. 313.

[4] Fauvel, "Sur la bronchite capillaire".—*Mémoires de la Société Médicale d'Observation*, t. ii, p. 561-573.

aspect. Still, even then there are many instances of "catarrhal" bronchitis where flakes of fibrine and pseudo-membranes are found in the midst of the muco-purulent expectoration.[1]

Besides, at the time when the disease is as yet called asthma, the bronchial mucous membrane is often the seat of destructive processes, which profoundly modify its secretion. There are not only superficial excoriations (catarrhal ulcers), but, in bronchiectasis more especially, a necrobiosis extends to the bronchial muscles,[2] and may successively involve even the different layers of the bronchial wall, so as to convert it into detritus.[3] In the sputa of the cases that Leyden describes as asthma, he always found tubular, contorted structures, which seemed to consist of a substance resembling coagulated mucus. Their surface exhibited an obliquely spiral delineation, and was studded with mucous corpuscles. A narrow homogeneous-looking thread ran along the axis of each of those structures, and terminated with a few spiral twists.[4] Those formations seem to be exfoliated glands. They were seen in the case of a patient who applied at the Victoria Park Hospital in December 1872. The patient, aged 34, a tailor by trade, was a native of Poland, but had been resident in this country for more than twelve

[1] Rilliet and Barthez, *Traité des maladies des Enfants.* Paris, 1853, t. i, p. 410. [2] Thierfelder, *Atlas*, Plate iii, fig. 2.
[3] Rindfleisch, *Lehrb.*, etc., p. 355. [4] *Loc. cit.*, p. 349.

years. At the age of 16 he had typhoid, and at 21 he had malarial fever, which lasted for one year and six weeks. Since then he had been frequently subject to colds, but did not suffer in any way so as to be unable to follow his employment. In 1871 he noticed a glandular enlargement in the right axilla, and similar swellings soon appeared in the left axillary, and in the cervical and inguinal regions. For the last six weeks he had nocturnal attacks of dyspnœa, which aroused him towards midnight, and continued with unabated intensity for hours, until, after a very painful cough, he expectorated a quantity of a greyish white and very viscid sputum.

The patient is of middle height and well built. His complexion is very dark, and his visible mucous membranes are very anæmic. He is greatly disfigured by a chain of glandular enlargements along the sterno-mastoids, varying in size from that of a chestnut to that of a small orange. They were moderately hard, movable, and irregular in shape. The glands in the arm-pits were much larger. The spleen was not enlarged, and repeated examination of the blood shewed no increase of white blood corpuscles. The thorax was broad, but flat, and its antero-posterior diameter was rather contracted. Respiration was abdominal, and not accelerated. Percussion of the thorax gave on both sides, front and back, a tympanitic note, and there was very feeble respiration, especially so on the

right side. The heart was covered by the lungs, its sounds were feeble but clear. The pulse was small, soft, and frequent.

The sputa were about four tablespoonfuls in quantity. They were of a greyish-white colour, embedded in a frothy mucus, and very viscid, so that they could be handled with knife and scissors. Thrown into water

FIG. 1.

FIG. 2.

FIG. 3.

they exhibited band-shaped masses of a pearly whiteness, and small pellets of the same colour. The former consisted of pus corpuscles, arranged in rows, and contained as it were in a fibrillar stratum. Besides large quantities of woollen and cotton fibres, and of what seemed to be zoogloea masses, there was an abundance of pavement epithelium, the octahedral

crystals (fig. 1) repeatedly mentioned, either entire, or the fragments in close apposition, the structures above described (fig. 2), and, apparently, casts of the smallest bronchioles (fig. 3), resembling almost those of the renal tubes, and studded with epithelium.

The origin and the persistence of the dyspnœa may thus be readily accounted for. The exudation itself causes obstruction of the bronchi, and the freedom of respiration can be re-established only when the obstacle is removed. But not all the sputa that are formed in the course of an inflammation are expectorated at the same time. A quantity of them remain behind, firmly adhering to the bronchial wall. So long as they are in the larger tubes, they do not interfere with respiration. The superficial breathing produces no stenotic sounds, and as the current of air is not sufficiently strong to separate the plug from the mucous membrane, there are consequently no rhonchi. But when the tenacious mucus gradually softens, it loses its adhesion to the surface, and may then be readily detached from this by brisk respiratory movements, such, for instance, as laughing. In that case, following its own gravity, the mucus falls downwards and occludes a portion of the lungs. Its separation and impaction are greatly facilitated during sleep, for here respiration is slow but deep; and in the recumbent posture, moreover, the smaller bronchi form, with the larger, an inclined plane,

as it were.[1] Those portions of mucus act like ball-valves; they descend and rise with inspiration and expiration; and not unfrequently they are, by the efforts at expulsion, forced beneath the cartilaginous plate, which projects like a spur, at the place where the bronchi subdivide. The dyspnœa lasts as long as the obstruction, and its intensity is directly proportional to the number and the calibres of the occluded tubes. Relief is obtained only when the obstacle is removed, either by expectoration of the mucus, moulded in the shape of pellets, or by its displacement into a larger bronchus, where for a time it is harmless.

III. *Stenosis of the Bronchi.*—The term stenosis is generally applied to the obturation of the bronchi, which is chiefly produced, not, as in the cases hitherto described, by a fluxion or inflammation of the mucous membrane, but by pressure, internal and external. In some instances the stenosis, with its consequences, is only one of a series of symptoms, and amongst these comparatively unimportant, as, for instance, in aneurism of the thoracic aorta. In others, again, the intermittent attacks of dyspnœa are the sole and most conspicuous results of such pressure, and thus tally with the definition of asthma as above given. The stenosis may be caused by

1. *Foreign Bodies.* Foreign bodies of various sizes and of different kinds may, unperceived, pass the

[1] Traube, *Gesammelte Abhandl.*, etc., Bd. ii, p. 17-23.

larynx, and penetrate to the bronchi. This occurs most frequently from inattention to the act of swallowing, or when food or other articles are present in the mouth whilst deep inspirations are taken, as during coughing and laughing. Children seem to be most liable to such accidents. Amongst the 170 cases collected by Aronssohn[1] there were 95 children under 10 years, and 16 at the age of from 10 to 15 years, so that more than 65 per cent. of all cases were children under 15 years. Professor Henoch, of Berlin, recently reported the case of a girl, 9 years old, who was brought to the hospital suffering from intense dyspnœa and cyanosis. She was perfectly well until the previous evening, but became restless during the night, and towards the morning the present symptoms manifested themselves. The physical examination of the chest showed the organs to be healthy. On her return home from the hospital, she began to vomit large quantities of undigested food, among which were specially noticeable *pieces of a hard-boiled egg*, which, as subsequently ascertained, she had *hastily swallowed the night before*. After the vomiting she had a good night's rest, and the next day the freedom of respiration was completely restored.[2] Professor Henoch thinks that the hard egg

[1] *Des corps étrangers dans les voies aériennes.* Strasbourg, 1856, pp. 64-68.

[2] "Ueber Asthma dyspepticum".—*Berl. Klin. Wochensch.* No. 18, 1876.

irritated the gastric filaments of the vagus, and caused a bronchial stricture. But it is at least as likely that the dyspnœa was caused by a bronchial obstruction.

It appears that some emphysematous are greatly exposed to that cause of the dyspnœa, on account of the uniform dilatation of the larynx and trachea which accompanies the dilatation of the bronchi and alveoli.[1] Patients of this class are frequently seized with an attack when they are spoken to during meals ; but they remain free from oppression at the time when their attention is not diverted from the business in which they are engaged. Hamburger lately recorded a very instructive case, which, although its symptoms differ from those peculiar to asthma, yet bears upon the present question. An old farmer complained of indefinite ailments, which the doctor whom he consulted attributed to some derangement of the nervous system. The patient's only daughter had recently got married, and it was supposed that his indisposition arose from a feeling of loneliness. Various means were tried, but none brought relief. As the patient constantly grew worse, a visit to his daughter was proposed, with the view of remedying the evil at its root. But the result did not answer the expectations. Shortly after his arrival at his daughter's house the patient became worse, so much so that he was forced even to keep his bed. When Hamburger was sent for, the case seemed

[1] Rokitansky, *op. cit.*, Bd. iii, p. 6.

to be one of cerebral hæmorrhage. But it struck him that the right side of the chest lagged behind in the respiratory movements, and though the percussion was perfectly normal, there was nevertheless complete absence of respiratory murmur. There was no evidence, nor even a suspicion, of a tumour compressing the main bronchus of the right side. The presence of a foreign body in it could not be so readily excluded, and this view gained ground on remembering the fact that a cause which produces dyspnœa in the adult, leads to a comatose condition in the aged. After much hesitation Hamburger at last decided to administer an emetic. He gave instruction that if vomiting took place, the matter should be kept for his inspection. In a few hours he returned. But great was the surprise when the patient, whose fatal end, it was feared, might be accelerated by the emetic, was sitting up in bed, overwhelming his deliverer with gratitude. The sputa contained a green pea, enormously swelled with moisture. The patient remembered that on gathering peas he had eaten some, but he had never noticed that one had entered the air passages.[1]

2. *Compression.*—Mediastinal tumours of various descriptions are the most common cause of bronchial compression. But though accompanied by attacks of asthma, they do not properly come within the limits here assigned to asthma for this reason, that the origin

[1] *Berlin. Kl. Wochensch.*, 1873, Nos. 28 and 29.

of the dyspnœa in those cases is readily discernible from the physical signs and the other attendant symptoms. It is different, however, with regard to the simple hypertrophy of the bronchial glands. Participating as they do in all the changes that take place in the surface or substance of the lungs, their volume increases when an abnormally large quantity of exudation is pressed into the radicles of the lymphatic vessels. In the catarrhal pneumonia of measles and whooping cough, the inundation of the pulmonary parenchyma with serum and white blood corpuscles always leads to a considerable enlargement of the bronchial glands.[1] But though this enlargement diminishes with the cessation of the inflammatory process, yet it seldom entirely disappears. The glands which accompany the bronchi to their third and fourth subdivisions are normally of the size of a lentil or of a pea ;[2] but especially in scrofulous patients, they are readily enlarged by even the slightest irritation to the size of a bean or of a hazel-nut. Cruveilhier repeatedly found such glandular hypertrophy deep in the substance of apparently healthy lungs.[3] Thus the bronchial tubes are flattened without being actually obliterated. Yet every fluxion or inflammation, while diminishing the calibre of the air tubes, simultaneously

[1] N. Gueneau de Mussy, "Etudes cliniques sur la Coqueluche". —*L'Union Médicale*, Nos. 81 and 85.

[2] Becker, "De glandulis thoracis lymphaticis atque thymo." D. J. Berol, 1826.

[3] *Traité d'Anatomie pathologique*, t. iv, 1862, p. 640.

increases the size of the lymphatic glands, and thus produces a complete obliteration of the bronchi. For a time this glandular enlargement was supposed to be peculiar to childhood and infancy, and the intermittent dyspnœa at that period of life was correctly ascribed to it;[1] but recent researches have shewn that the asthmatic seizures of adults may also arise in the same way.[2]

[1] Rilliet et Barthez, *op. cit.*, t. iii, p. 647, *et seq.*

[2] Noel Gueneau de Mussy, "Adenopathie bronchique".—*Clinique Médicale*, tome i. Paris, 1874, p. 569 *et seq.*

CHAPTER V.

CLINICAL HISTORY.

It is in accordance with its nature as a symptom of chronic pathological processes that asthma only gradually assumes its characteristic clinical features. Slow as their development is, it is nevertheless attended by certain phenomena, which, if correctly interpreted, plainly indicate the approach of the disease. The significance of these premonitory signs, however, is at first apt to be overlooked, and thus it often happens that the typical attacks of dyspnœa set in unexpectedly, taking the patient quite by surprise.

In those cases in which asthma originates from whooping cough and measles, it is in fact merely the direct continuation of the catarrhal pneumonia. Long after the febrile symptoms have completely subsided, the cough and the dyspnœa, which have equally abated through the day, return at night with their former intensity. These true paroxysms of asthma, or rather this early manifestation of it, are at the time rightly ascribed to the inflammatory affection, the influence of which is as yet remembered. But when the nocturnal dyspnœa also ceases, recovery is now supposed to be complete. The freedom of respiration and the nega-

tive results of physical examination of the chest give rise to no suspicion of the irremediable injury which the lungs have sustained by the previous pneumonia. Yet the friends of the patient are less readily satisfied as to his present condition. They notice with special concern that his breathing continues to be laborious after the slightest exertion, and that during sleep it is often so noisy as to be audible even in an adjoining room. Indeed, their apprehensions are soon realised; the dyspnœa returns without known or appreciable cause, but subsides, after lasting several hours, in the same mysterious manner. In some instances a considerable time, during which respiration is in no way impaired, elapses before the appearance of the typical paroxysm, so that its antecedents are almost forgotten. In either case, however, the patient is henceforth a confirmed asthmatic.

The development of the disease is much more insidious in the atrophous forms of emphysema and bronchiectasis. As this process does not at first implicate the surface of the bronchi, there is consequently little or no cough or expectoration. The conspicuous susceptibility of the bronchial lining to hyperæmia is merely looked upon as a proneness to "colds". No notice is taken of it, and when asthma declares itself, those patients expressly deny having ever been subject to an affection of the chest; yet, even under these circumstances, asthma rarely fails to give fair warning

of its approach. Apart from numerous and indefinite ailments, all proceeding from and connected with the disease of the lungs, there are certain symptoms particularly significant in that respect.

Some patients suffer for a long time from attacks of nightmare. Frequently their sleep is disturbed by a painful oppression at the chest, which they, in their semi-conscious condition, interpret in a fanciful manner, imagining themselves engaged, for instance, in a combat with some monstrous creature. With the first deep inspiration, however, on waking, the oppression ceases, and the abnormal sensation is regarded as a dream, due to an indigestible supper. Advice is seldom sought for it, as being a matter of no consequence ; all that is necessary, it is thought, is to act according to the adage, "*Ut sis nocte levis, sit tibi cœna brevis*". Subsequently the patients are wont to relate this circumstance in order to prove their alleged nervousness. Yet the attacks of nightmare gradually increase in intensity, and ultimately develope themselves into asthmatic paroxysms, from which indeed they only differ in degree.

Others, again, exhibit an abnormal sensitiveness of the respiratory surface and of the skin. They notice that in the summer and autumn, from May till September, they are apt to suffer from uncontrollable fits of sneezing, which, by no effort of imagination, can be ascribed to "colds". At the same time the irritation

generally spreads to the conjunctivæ, to the fauces, and to the soft palate, producing a congestive swelling with its consequences. There is an almost intolerable itching, and the sensation as if those parts were sprinkled with cayenne pepper. The skin, and especially those portions of it exposed to the air, is similarly affected. The irritation, which is not relieved by scratching, is promptly followed by a herpetic eruption. These symptoms are peculiar to "hay-fever", and are doubtless caused by pollen grains in the air. But, however much opinions may differ as to their causation,[1] the connection of those sympoms with asthma is so well established that they have generally been recognised as the precursors of the disease.[2]

Such train of symptoms frequently appears and disappears for an indefinite period before the asthmatic paroxysm manifests itself. In typical cases the dyspnœal seizures generally occur at night, and quite unexpectedly, the warning of their approach being as yet imperfectly understood. The patient goes to bed in his usual state of health, paying no attention to, or unable to account for, a feeling of fatigue and of drowsiness, with an aching pain in, or a weariness of, his

[1] Eulenburg thinks the fits of sneezing may be caused by the first fœtal movements, because a woman suffered from them at the fourth month of her pregnancy.—*Lehrb. d. function. Nervenkrankh.* Berlin, 1871, p. 672.

[2] Ferber, *loc. cit.*; Trousseau, *loc. cit.*

limbs. After a few hours' sleep, generally disturbed by dreams, he is, towards midnight or early morning, suddenly roused by a great oppression at the chest. He starts from his sleep in bewilderment, not knowing at first whether all this be not a dream. But the threatening suffocation soon convinces him of the terrible reality. He leaps from his bed, rushes to the door or the window, and exposes himself, regardless of all the dreaded consequences, to the cold night air, but finds, to his amazement, that the air does not enter his lungs beyond a certain distance. He feels as if his bronchial tubes were constricted. There is a tightness across his chest, as if a cord were firmly wound round it. His life seems in danger unless this constriction be removed; and all his efforts, therefore, are directed to dilate the thorax. In order to more effectually attain this object, every muscle concerned in aiding thoracic play is brought into requisition. To facilitate their action, various attitudes are assumed. The patient stands erect, with the head thrown backwards, seizing some solid object to give greater vigour to his efforts; he sits in his chair, on the arms of which his elbows are fixed or his hands are planted on his knees; he leans forward on a table, holding his head between his hands, or sits across a chair, supporting his head on the back of it. The imperious desire for air engrosses all his energy and attention, so that he is unable or unwilling to speak or even to move his

head in answer to questions that may be put to him. Yet, in spite of the straining and tugging of the muscles, the respiratory wants remain unsatisfied. The short and painful cough, by which he is incessantly tormented, raises but a small quantity of a frothy substance, without dislodging the obstacle which he distinctly perceives in his air-passages. His anxiety increases; death seems imminent. With avidity he tries all remedies suggested to him, but their inefficacy only renders him impatient and adds to his terror.

At this stage of the attack, the asthmatic presents a most distressing aspect. The attitude at first assumed is carefully preserved. The face is expressive of the greatest anxiety; pale at the commencement of the attack, it soon becomes dusky and cyanotic. The perspiration stands in beads on his forehead, or even runs in drops down his face. The eyes are wide open, staring, projecting, watery, or suffused. The alæ nasi work powerfully. The mouth gapes with each inspiration. The shoulders are drawn up to the head, which seems buried between them. The thorax is immovable; distended to its utmost limits, it cannot follow the forcible traction of the respiratory muscles. Respiration is noisy, and accompanied by a wheezing that may be heard at a distance. The hands and the feet are icy cold. The temperature of the body is greatly diminished, and no amount of friction can restore the natural warmth of the extremities.

Disturbances of circulation necessarily accompany this dyspnœa. The arteries are imperfectly filled, and the pulse is generally frequent and so small as to be hardly perceptible. Sometimes it grows distinctly irregular, although the patient be free from heart disease. The venous system is, on the contrary, gorged, so that the jugular veins project like large black cords. The venous congestion of the brain may give rise to headache, vertigo, and, according to Bamberger and Lebert,[1] also to clonic contractions of the various muscles of the body; but Dr. Walshe has never known convulsions to occur even in the worst specimens of asthmatic paroxysms.[2]

A profuse diuresis of a pale and limpid urine at times accompanies the outset of, and persists throughout, the attack. Generally, however, the urine is, in the height of the dyspnœa, diminished in quantity, high-coloured, and loaded with lithates, but becomes at the end of the paroxysm more abundant and clear. Dr. Ringer observed in one case that during and immediately after the fit, there was a considerable deficiency of urea and chloride of sodium.[3] Occasionally the urine contains albumen and casts.

The physical signs are generally the following :—In the height of a paroxysm all the muscles accessory to

[1] *Op. cit.*, p. 425.

[2] *Diseases of the Lungs*, p. 547.

[3] Parkes, *op. cit.*, p. 319.

respiration may be seen to be firmly contracted. As regards inspiration, the utmost activity is displayed not only by the sterno-mastoids, which project like rigid cords, but also by all the other muscles that pass from the head to the shoulders, to the clavicles, and to the ribs. With each such act the hyoid bone is forcibly drawn down by the muscles inserted between it and the sternum; while the trapezii and levatores anguli scapulæ raise in their turn the shoulders almost to the ears, and thus indirectly assist in elevating the ribs. The diaphragm—the most important muscle of inspiration—seems to be even in a state of tonic contraction, an appearance in a great measure due to its limited excursions in consequence of the distension of the lungs. In the same degree expiration is reinforced by the activity of all the muscles subservient to it, as the rectus, the obliqui and transversalis abdominis, the quadratus lumborum, the latissimus dorsi, and others.

In spite of the forcible activity of all these muscles, the ventilation of the lungs is imperfect. Although the capacity of the thorax is enlarged to its utmost limits, yet this extreme distension remains unaltered during expiration, so that the chest seems almost motionless. The result is that the interchange of gases is considerably reduced, and becomes so scanty indeed, that in order to maintain life, even at its lowest ebb, all the oxygen of the inspired air is absorbed; for

while under normal conditions 100 volumes of the expired air contain about 15·4 volumes of oxygen, discharged as apparently superfluous, no oxygen was found in the two analyses by Heurtaux, in which the carbonic acid was increased to from 6·8 to 11 volumes per cent.; nitrogen was represented by from 93·2 to 89 volumes per cent.[1]

The frequency of respiration is generally diminished, at times it is reduced as low as 10 or 9 acts in a minute.[2] This reduction is due to the prolongation of expiration, which always takes place in cases where the obstruction occupies the bronchi.

While watching the respiratory movements, the deformities of the thorax and of the spine cannot fail to attract attention. There are but few patients in whom the configuration of these parts is completely normal. In some, the chest may be relatively too large, probably owing to a morbid growth of bone in the costal cartilages;[3] or it may be of the paralytic type, which at an early period of the disease is often concealed by a large quantity of subcutaneous fat, but becomes more conspicuous when, after the persistence

[1] Quoted by Germain Sée, *loc. cit.*, p. 615.

[2] Guastalla, "Ein Fall von nervösem Bronchialasthma" (*Jahrb. f. Kinderkrankheiten*, vii, 210, 1874), observed the frequency of respiration as 60, and Politzer, *Ibid.*, iii, 377, 1870, as 50 per minute.

[3] Freund, *Beiträge zur Histologie d. Rippenknorpel*, 1858; and *Der Zusammenhang gewisser Lungenkrankh.*, etc. Erlangen, 1859.

of asthma, emaciation occurs. Others, again, are pigeon-breasted; that is, the lower part of the sternum projects in the shape of a ridge, at the sides of which there are two hollows corresponding to the costal cartilages from the seventh to the tenth. A greater variety of deformity is observable in the spine,—from the absence of its natural curves to the extreme degrees of kyphosis, lordosis, and scoliosis.

Percussion shows the descent of the lungs by two or more intercostal spaces, so as to completely fill up the sinus pleuræ. The note which it produces is over all parts of the thorax hyperresonant, and accompanied by a tympanitic ring. This sound remains unaltered within almost the same area during expiration, in consequence of the permanent inspiratory position of the organ.

The vesicular murmur is either very feeble or completely suppressed. Instead of it, there are in both phases of respiration sonorous and sibilant rhonchi, so loud as to be audible even at a distance, and with their greatest intensity generally at the bases. In some cases these sounds are apt to change their site. The complete silence at one spot, and the loud wheezing at another, may even, during observation, give way either spontaneously, as it seems, or by a deliberate and deep expiration, to the normal vesicular murmur; or the vesicular murmur may suddenly disappear and be replaced by the sonorous rhonchi. Such

changes can be produced only by a plug of mucus, which is moved to and fro by the respiratory current. There may be occasionally crepitation. This, however, ceases when, by means of a powerful cough, a quantity of a watery sputum is expectorated. Such crepitation occurs in attacks connected with Bright's disease or other affections of the urinary organs, accompanied by suppression of urine.[1]

The physical exploration of the heart gives but imperfect results. The cardiac dulness cannot be distinguished by means of percussion, because the organ is entirely overlapped by the distended lungs. The cardiac impulse is diffuse and visible beneath the xiphoid cartilage, but it is so feeble as to be almost imperceptible. The rhythm, regular at first, grows intermittent if the attack is prolonged. The sounds are, in the absence of organic lesions, clear at the commencement of the paroxysm, but subsequently they either become less distinct, or are altogether drowned by the noise produced in the lungs.

All these symptoms may either rapidly—within a few minutes—or gradually attain their maximum intensity. Their duration also greatly varies—from a few minutes or hours to several days, weeks, or even months.[2] Not infrequently the attack subsides as quickly as it appeared, so that within half an hour the

[1] Niemeyer-Seitz, *op. cit.*, p. 91.

[2] Trousseau, *op. cit.*, p. 440.

dyspnœa may have ceased, and the patient may be as well as ever. In the majority of instances the paroxysm lasts for several hours ; it commences towards midnight, and abates with the break of day. In severe cases, when the attack is protracted during weeks, comparative intermissions alternate with exacerbations of the symptoms ; inasmuch as a certain freedom of respiration for some hours of the day is followed at night by dyspnœa in all its intensity, so that the disease then appears to consist of a series of paroxysms. Now the most trifling cause may serve to prolong or to rekindle the attack. Aware of this fact, the patient on that account abstains from even the slightest movement, and persistently maintains the attitude he has once chosen, and from which he promises himself the most speedy mitigation of his suffering. Although in many cases the appetite is noways impaired, yet the patient does not dare to eat. The business of his life seems centred in the one object —to rid his bronchial tubes of the obstruction, and to supply his respiratory wants. Fainting with hunger, he subjects himself to starvation rather than for a minute to neglect his respiratory efforts or to do anything that might possibly increase his dyspnœa. For instinctively or by experience he knows that food would only aggravate his present trouble.

At last the severity of the attack abates, and it usually does so coincidently with the appearance of

expectoration. The harsh and painful cough now becomes looser; the dry and wheezing sounds are replaced by moist râles; and the sputa, instead of being scanty and frothy as hitherto, are now more abundant and also altered in quality. The quantity and the composition of the sputa vary according to the nature and the intensity of the paroxysm. Thus a very transient seizure may pass off without any expectoration. A more lasting dyspnœa may terminate with a sparing amount of a frothy substance, consisting chiefly of water, of alkaline salts, and of a few mucus corpuscles. Occasionally the sputa present all the characteristics described by Laennec as peculiar to the "pituitous catarrh". They resemble in appearance a solution of gum arabic, or of white of egg; they are very viscid, and contain large quantities of albumen. At times a few streaks of blood may be mixed with the expectoration; or a sanguinolent foam is all that is brought up. It rarely happens that pure blood is coughed up, and when this occurs, the quantity is generally very small—a teaspoonful or so. Yet Hyde Salter has known it to amount to a profuse hæmoptysis, from half a pint to a pint.[1]

In inveterate cases of bronchiectasis the attacks terminate with very abundant muco-purulent sputa, which, having undergone decomposition within the dilated tubes, possess often a penetrant and nauseous odour.

Frequently, the expectoration consists of only a few pellets of a viscid, grey mucus. The greater quantity of the bronchial secretion is then retained within the lungs, and merely displaced into larger tubes, where its presence is harmless for a time.

In severe and protracted cases the sputa present all the characters previously described[1] as peculiar to fibrinous (croupous) bronchitis. The green colour which the flocculent and granular matter occasionally presents, is due to the presence of spores.[2]

When the paroxysm is at an end, the patient feels greatly exhausted; a few hours' sleep, however, refreshes him so much that, on waking, nothing but a slight pain in the muscles, the wheezing respiration, and the persistence of the expectoration, remind him of the agony through which he has passed. Although his breathing is now greatly improved, the dyspnœa is not yet entirely gone; but the remembrance of his former sufferings renders a state—unbearable perhaps to others—to him, one of ease and comfort. Recovery, however, soon becomes complete.

If the disease has so far declared itself, the attacks never fail to return; and the patient must thus look upon himself as asthmatic for life, unless his dyspnœa depend upon the presence of a foreign body in his air-

[1] *Conf.*, p. 165, *et seq.*; Lefevre, *op. cit.*; Hyde Salter, *op. cit.*, p. 337; Robertson, *Americ. Journ.*, etc., 1841, p. 252.

[2] O. Rossbach, "Ueber eine neue Art von grassgrünem Sputum." —*Berl. Kl. Wochensch.*, No. 48, 1875.

passages, in which case it may permanently subside after the expulsion of the obstructive agent. In all other cases, the recurrence of the exciting causes is invariably attended by the reappearance of the asthmatic seizures. These are, at an early period of the disease, generally separated by long but indefinite intervals, which, however, with the progress of the anatomical changes of the lungs, gradually shorten, until at last the dyspnœa becomes permanent. Nor is there an intrinsic periodicity independent of external circumstances; but the attacks occur at all times of the day or night, whenever circumstances are favourable for their production. If, nevertheless, they occasionally seem to possess a certain regularity in their appearance, this is due to the regular exposure of the patient to, as well as to the nature of, the exciting causes. The influence of the latter is illustrated by "hay-asthma", which occurs during the summer and autumn, because the air contains at those seasons only pollen in sufficient quantity and of the necessary vitality. But, as other substances, such as, for instance, ipecacuanha powder, mustard, and various fungi or their spores, have the same effect as pollen (*i.e.*, fluxionary hyperæmia of the parts with which they come in contact), the same symptoms may be observed at all times of the year.

In the intervals between the attacks, respiration is generally undisturbed. With proper care as to fresh

air and out-door exercise—of the utility of which their instinct advises them—asthmatics remain singularly exempt from cough, much more so, indeed, than many healthy persons, who discover, in every perceptible current of air, the source of a fatal illness. Yet, even at an early period of the disease, the destruction of the pulmonary capillaries and the changes it produces in the venous system are felt throughout the entire organism. There is the disposition, repeatedly alluded to, on the part of the skin and of all mucous membranes, to catarrhal affections. Nothing, however, causes asthmatics so much suffering as their dyspepsia. They generally possess a good appetite, and, under the erroneous impression that the more food they consume the more strength they gain to cope with their malady, they overload their digestive organs, and thus increase the evil. It is difficult, and often in vain, to persuade them that they derive benefit only, not from what they eat, but from what they assimilate.

All these symptoms are necessarily aggravated by every fresh impediment of the respiration. The imperfect decarbonisation of the blood manifests itself by headache, vertigo, or drowsiness, which may be so great that the patient cannot possibly keep awake; by a dulness of perception, and an irritability of temper, which the friends are the first to notice; by constant yawning, pains of the limbs, and pain at

the epigastrium. If the stomach has been overloaded, the result is a flatulent distension of the intestines, and also constipation. These phenomena are promptly followed by an attack, and, as a painful experience soon teaches the patient their significance, he learns to look upon them as the "aura" of his asthma.

Gradually, the original disease of the lungs tends to its complete development, to which the attacks, in their turn, materially contribute. The intervals of the respiration become shorter, and, instead of complete remissions, there are now only abatements of the dyspnœa. The disease that might at first be mistaken for a purely nervous affection, declares itself at last as phthisis, emphysema, or heart disease.

CHAPTER VI.

DIAGNOSIS AND PROGNOSIS.

Diagnosis.

THE diagnosis of asthma is mainly dependent on the physical signs during an attack. The distension of the chest in all its diameters; the prolonged, laboured, and ineffectual expiration; the hyper-resonance on percussion of especially the bases; the suppression of the vesicular respiration and the presence of sonorous, and sibilant rhonchi; these are, in so far, characteristic of the disease, as they indicate an obstruction of the bronchi. No other form of paroxysmal dyspnœa presents a similar series of objective symptoms, so that, by attention to them, asthma may be readily distinguished from

1. Angina pectoris.
2. Obstruction of the larynx.
3. Paralysis of the posterior crico-arytenoid muscles.
4. Spasm of the diaphragm.
5. Paralysis of the diaphragm.

1. Angina pectoris rarely occurs for the first time before the age of fifty, while asthma, on the contrary, most frequently commences in childhood, or at puberty.

Its dominant symptom is pain of a gnawing or lancinating kind, which is situated beneath and at the left side of the sternum, and thence radiates to the spine, to the left side of the neck, and to the left arm. The sensation which asthmatics experience during the attack, is one of fulness of, or of tightness across, the chest, rather than one that amounts to actual pain. Dyspnœa is by no means an essential feature of angina, and, if it is present, it is never so intense as in asthma. Moreover, in the former, the chest is not distended, and the respiratory movements are very superficial, on account of the pain which they occasion.

2. In obstruction of the larynx, from whatever cause, the physical signs are just the opposite to those peculiar to asthma. Notwithstanding the vigorous action of the inspiratory muscles, little air enters the air passages through the obstructed larynx; the lungs are, therefore, retracted so far as the chest walls permit, while the yielding portions of the thorax are depressed by the external atmosphere. The insertion of the diaphragm is marked by a deep furrow. The frequency of respiration is here also, as in asthma, greatly diminished; but this reduction is due to the prolonged and laboured inspiration, instead of expiration, as in the latter. Inspiration is accompanied by a noise which is produced in the larynx, and is most audible at its place of origin; but it is faintly conducted towards the bases of the lungs. In asthma,

on the contrary, the wheezing is generally loudest at the bases, but diminishes in intensity on ascending towards the larynx. Thus, while the dyspnœa of asthma is of the expiratory, that of laryngeal obstruction is of the inspiratory type.

3. In paralysis of the posterior crico-arytenoid muscles, there is always a more or less continuous dyspnœa, which, on laryngoscopic examination, will be found to be caused by the close approximation of the vocal cords, so as to form only a narrow chink for the passage of the air.

4. A tonic spasm of the diaphragm is as yet chiefly known from the experiments of Duchenne.[1] In the two fatal cases in which it has been clinically observed,[2] there was, besides the dyspnœa, tetanus of all the muscles of the trunk, and loss of consciousness, in the height of the attack; symptoms, the presence of which sufficiently distinguish it from asthma.

In clonic spasm of the diaphragm (hiccup, singultus) respiration is greatly accelerated. The inspirations are rapid, forcible, and accompanied by a peculiar noise, produced in the larynx and reinforced in the fauces. The lungs, however, are imperfectly filled with air, and the yielding portions of the thorax are depressed.

5. Paralysis of the diaphragm, an affection of very rare occurrence, manifests itself by the inversion of

[1] *Conf.*, p. 35. [2] Valette and Bamberger.

the normal respiratory movements. The epigastrium and hypochondrium, instead of projecting during inspiration, are forcibly drawn inward, because the paralysed diaphragm follows the traction of the elastic lungs. The breathing is greatly accelerated to from forty to fifty in a minute, and there is absence of all adventitious sounds, if neither laryngeal nor bronchial affections complicate the attack.

Significant as are the physical signs in respect of the bronchial obstruction, they yet afford but little evidence as to the nature of the obstacle, by which, alone, the different forms of asthma are distinguishable from each other. Information on this subject is obtained only from the circumstances attending each case. But this, although a matter of great practical importance as regards the treatment, is often one of considerable difficulty, especially when nothing is known of the patient, and when in the presence of his acute suffering, there is hardly time for an elaborate inquiry of any kind. Happily, however, there are generally some points indicative of a certain track, the pursuit of which may lead to the desired end.

Thus the presence of the viscid mucus within the bronchi may be inferred from the *râles* changing their site while under observation. The rapid succession of complete silence, of vesicular murmur, and of wheezing sounds, cannot be accounted for in any other way.

Neither hyperæmia nor compression of the air tubes can be so suddenly produced and removed; and the presence of foreign bodies may be also excluded, since these phenomena simultaneously appear at several parts of the thorax. During the attack the sputa are scanty; but the patients know from experience that their dyspnœa ceases with a more or less abundant expectoration.

The occurrence of the dyspnœa soon after a meal or in consquence of constipation, is suggestive of hyperæmia of the lungs. In that case, the intestines are so distended with gas, that the diaphragm is pushed upwards to the fourth or even third rib. The respiratory murmur is feeble or inaudible, and, occasionally, a loud wheezing is heard. The heart is displaced and the pulse is full and bounding.

If the dyspnœa is immediately preceded by fits of sneezing, it may be suspected that there is in all probability a fluxionary hyperæmia of the lungs, produced by the inhalation of mechanical irritants floating in the air. In the summer and autumn the presence of pollen grains in the atmosphere is the most frequent cause of the disease, so that the patients chiefly suffer in the open air, or at places to which those bodies are carried by the wind. The dyspnœal attacks are not unfrequently accompanied by itching of the skin, by a herpetic eruption of the face, and by a burning sensation of the fauces. There is, moreover, a short and pain-

ful cough and a frothy expectoration, in which pollen may be detected on microscopic examination. Moreover, the history of the case shows that on former occasions these symptoms subsided, and that immediate relief was obtained when the patient removed to the crowded neighbourhood of large cities, the air of which is supposed to have a special curative effect upon asthma.

Œdema of the lungs occurs most frequently in hydræmic patients and in those suffering from Bright's disease. It generally betrays itself by great cyanosis, by a feeble and almost empty pulse, and by a weak action of the heart. The rhonchi are, as a rule, of a moist character, and the sputa are copious, frothy, and at times tinged with blood. Towards the end of the attack there is generally profuse diuresis. In the interstitial œdema, which accompanies renal affections, there are but few and scattered rhonchi, because no or very little fluid transudes into the bronchi and alveoli. Albumen and renal casts are frequently found on examination of the urine.

Embolism of the pulmonary artery, notwithstanding its frequent occurrence and the attention with which its pathology has been studied, as yet eludes detection. Its presence, however, may be suspected if, in the feeble and cachectic patient, or in those affected with disease of the heart, especially dilatation of its right side, and with varicose veins, the dyspnœa, which suddenly arises and without any

premonition, rapidly assumes a great intensity. There are, moreover, all the objective symptoms of a pulmonary œdema, with this difference only, that, in one portion of the lung, probably corresponding to the obstructed arterial branch, there is a complete absence of vesicular murmur and of all adventitious sounds, while percussion produces a resonant note.

The simple enlargement of the bronchial glands, which accompanies hyperæmia and inflammation of the surface or substance of the lungs, is seldom of such extent as to be discernible by dulness on percussion of the manubrium sterni and the interscapula regions. But, as it nevertheless suffices to impede the access of air to the lungs, there is generally complete absence of all respiratory sounds, both after cough as well as after manual compression of the chest. These symptoms are generally observable on one side of the chest, whereas the other is the seat of the various catarrhal rhōnchi. Glandular enlargement in the cervical and supra-clavicular regions, corresponding to the obstructed side of the thorax, are of great diagnostic importance. Mediastinal tumours of a certain size are in general readily detected.

It rarely, however, happens that the asthmatic seizures are witnessed by a competent observer, and even busy practitioners have seldom, or never, an opportunity of exploring the chest of a patient labouring under a paroxysm. Advice is generally sought in

the intervals between the attacks, when the characteristic symptoms of these have completely subsided. In that case, the diagnosis can be based only on the consideration of the present state of the patient and on the history of the case. The seeming integrity of the thoracic organs, or the presence of chronic bronchitis and of emphysema, renders it highly probable that the impediment of respiration is situated with the bronchi, especially if the larynx is, on inspection, found to be free from disease or from tumours. The nature of the obstacle can be inferred only from the associated circumstances. In all cases in which the dyspnœa occurs for the first time at a comparatively late period of life and without such antecedents as whooping cough and measles, the urine should be repeatedly examined, even if a hypertrophy of the left ventricle or a great arterial lesion does not direct attention to the state of the kidneys.

Prognosis.

When asthma has once declared itself, there are, as a rule, irremediable changes of the pulmonary tissue, the nature and extent of which vary in different cases. A complete recovery is possible only in children in whom the disease is produced by a simple hypertrophy of the bronchial glands, as this enlargement occasionally subsides, and the irritability of the lymphatic system also diminishes towards puberty. But this favourable

issue is very rare, and there are few instances in which children have been known to "grow out" of their complaint.

Although a tendency to the recurrence of the dyspnœa thus remains for life, respiration may nevertheless be, and frequently is, quite undisturbed in the intervals between the attacks—a result that cannot be surprising, in view of the fact that perfectly sound lungs are a luxury, which only few are permitted to enjoy. The attention which asthmatics are prompted to pay to hygienic measures, gives them even a certain immunity from bronchial affections. So striking is this immunity, that Lebert, observing the safety with which asthmatics may expose themselves to all inclemencies of the weather, is tempted to believe that their disease affords them a protection against colds[1]; while Trousseau, who could not understand the predilection of the patients for fresh air, large rooms and open windows, considered it as "*une sorte de manie*" on their part.[2] Upon the whole it may be said that every asthmatic seizure is more or less a preventible accident, so that an efficient and suitable prophylaxis is almost tantamount to a cure.

The prognosis as to life is in general favourable. This is especially the case with the atrophous forms of the disease, *i.e.* emphysema. It frequently happens

that emphysema developes itself in infancy, as the immediate consequence of a catarrhal pneumonia, and yet the patient lives to the age of seventy and beyond, in spite of the intercurrent attacks of dyspnœa. The hypertrophic forms of the disease are, on the contrary, of more serious import. The interstitial pneumonia, which for a time escapes detection by means of physical diagnosis, manifests itself then only by the persistence of a copious expectoration in the intervals between the attacks. The symptoms of phthisis, however, soon become more apparent, and the disease generally terminates fatally in the prime of life. In these cases Trousseau speaks of a convertibility between nervous asthma and tuberculosis.[1] It is needless to add that the presence of cardiac affections renders the prognosis very unfavourable.

The appearance of asthmatic seizures for the first time at a more advanced period of life, is generally connected with either mediastinal tumours or with renal affection. Here, as a rule, the dyspnœa soon becomes continuous, and the course of the disease is then rapid and fatal. In rare instances, however, life may nevertheless be preserved and a certain freedom of respiration be restored.

However distressing the attacks may be, both to the patient and to the witness of his suffering, they seldom or never terminate fatally. Lebert reports only one

1 *Op. cit.*, p. 465 *et seq.*

case in which death ensued during the paroxysm, and even here it was, to a great extent, the fault of the patient, who disobeyed the strict injunctions not to evacuate his bowels, as the probable consequences of the straining were foreseen.[1] Generally the dyspnœa subsides more or less completely, after having lasted from several hours to several weeks. The return of the attacks rests chiefly with the patients themselves.

The course of the disease, however, may be favourably modified by therapeutic measures. Although asthma is generally looked upon as the most intractable of all diseases, yet this view is chiefly attributable to the manner in which the treatment has been hitherto conducted. Under the erroneous impression that the asthmatic tendency was not of the slightest consequence,[2] and that prevention was impracticable owing to the capriciousness of the disease, all efforts have on that account been directed merely to the mitigation of the paroxysms. That the blunting of the nervous system, the practice generally adopted, can have more than a temporary and palliative effect, is hardly to be expected. In asthma also it is necessary to meet all indications of the disease, and in doing so, there is every prospect that its treatment is at least as successful as that of every other chronic affection.

So long as the asthmatic tendency was supposed to be a mysterious derangement of the nervous system, there could obviously be no question as to the prevention of the disease. But, according to the view here adopted of its nature, an efficient prophylaxis, though, for various reasons, impracticable in some cases, appears by no means so in all of them. On the contrary, the fact that in the vast majority of instances, asthma is traceable to the pneumonia of whooping-cough and measles, affords just grounds for assuming that, by timely subduing the catarrhal inflammation, the subsequent disposition to the dyspnœal seizures may be either completely averted or greatly diminished. Whether this assumption will prove correct or not, is a matter of future observation. Meanwhile, although every case of catarrhal pneumonia is not necessarily developed into asthma, still, as this so often appears to be the consequence of the former, it is no superfluous prevention to guard against the contingency.

The precautionary measures required for the purpose merely consist in the judicious management of

those affections, in which the inflammation of the bronchi has the tendency to spread to the parenchyma of the lungs. This is not the place to inquire whether chemical irritants[1] or living organisms, floating in the air, are the causes of the complication; probably both are so concerned, inasmuch as the former supply the "phlogistic zymoid"[2] which developes the activity of the latter. Be this, however, as it may, certain it is that catarrhal pneumonia is most rife in ill-ventilated rooms, in which those agents are known to attain their greatest virulence. Hence, to protect the inflamed surface of the larynx and of the bronchi would appear to be the first and indispensable condition to prevent the extension of the disease. This, indeed, is merely the application to medicine of a practice to which modern surgery is mainly indebted for its successes; and could the diseased surface of the bronchi be as effectually protected as an open sore, the results would be, no doubt, as favourable in the one as in the other case.

With a view of carrying out that practice, attempts have been made to destroy, by inhalation of antiseptics, the organic substances contained in the inspired air, and those also accumulating upon the surface of the air-tubes; but in whooping-cough, in which the inhalations have often been tried, the results did not

[1] Bartels, *loc. cit.*

[2] Billroth.—Virchow's *Jahresbericht*, 1874, Bd. i, 352 *et seq.*

quite answer the expectations. More readily attainable, and, indeed, more beneficial, is the removal of the irritants from, or the prevention of their accumulation in, the air. So long as the patient suffering from whooping cough or measles is confined to his room, this should be effectually ventilated day and night. But if the weather and the state of the fever permit, he should spend the greater part of the day out of doors. Even a slight febrile movement is no contra-indication of his leaving the house; for not unfrequently the febrile movement is kept up by the vitiated atmosphere indoors. Unfortunately, parents, blinded by prejudice, often oppose such measures, and even part with the medical adviser who holds, what they conceive, such heterodox views. But their fear will be removed if they but remember that experience had long since shown that intractable cases of whooping cough were favourably modified by a "change of air", even if the place selected possessed no superiority of climatic conditions. That the beneficial effects are not attributable to the "change" as such, but to the free supply of fresh air, need hardly be mentioned.

In this way only is it possible to meet the requirements of the moment, and to obviate the more remote consequence; for there can be little doubt that the inflamed surface of the bronchi, protected against external irritants, and under the influence of an efficient

oxygenation of the blood, tends in those cases of itself to recovery. The exhibition of narcotics is, at the best, but of doubtful utility. Even if, by a sufficiently large dose of the drugs, the cough is allayed for a time, this, nevertheless, returns with its former violence when their physiological effect has ceased, unless the vitality of the patient is in the mean time exhausted. That it should be so, is obvious from the pathological conditions. Neither the inflammation of the mucous membrane, which is probably produced and maintained by peculiar fungi (Letzerich), nor the external irritants exciting the cough (Hauke), are in any way affected by sedatives; but both retain their unabated activity, however much the irritability of the nervous system may be reduced by treatment. As a rule, narcotics are even hurtful, in that they produce within and around the patient conditions that aggravate his disease. If proof were wanted, it is but necessary to watch a child suffering from whooping cough, in whom every effort of nature to expel the offending substances from the lungs is immediately suppressed by the inhalation of chloroform. The cyanosis, due to the obstruction of the bronchi, is increased by the means intended to relieve it; so that the treatment, if it does not hasten the fatal end, serves at any rate to cripple the patient for life.

B. *Treatment of the Disease.*

When asthma has declared itself, there are, as a rule, changes of the pulmonary tissues, which vary in nature and extent in different cases. But though those changes do not always betray themselves by adequate physical signs, they are, notwithstanding their latency or seeming insignificance, essentially the disease, and no plan of treatment, useless in the first place and specially directed to them, can be productive of permanent benefit. Indeed, the proverbial intractability of asthma is mainly due to the exclusive attention hitherto bestowed upon the dyspnœal paroxysms, at the expense of the organic lesions from which they arise. It is true those paroxysms are, for a time, the sole and always the most conspicuous symptoms, whereas the disease proper is quite compatible with perfect freedom of respiration ; and the inference may well be, as, indeed, it has been, drawn that if the paroxysms are relieved, the disease is rendered comparatively trifling. But experience has amply shown that this object is not readily attainable ; and even if it were so, still, as the asthmatic seizures are, in the vast majority of instances, avoidable accidents—it would be a truism to state—the prevention of their recurrence is more important than the mitigation of their severity. But no amount of attention to the exciting causes will protect against the attacks of asthma, unless the morbid

susceptibility to them be simultaneously diminished. The treatment of the disease alone promises to save the patient a vast amount of suffering. Nor is this the sole benefit that is likely to accrue from it. So long as the cardiac and pulmonary affections, which at a later period of asthma are readily recognised by their physical signs, were considered to be the consequences of the dyspnœa, the suppression of this was all that was wanted, although no one instance can be adduced to show that the desired result had been obtained in this way. But since those affections are known to be the terminal links in a chain of morbid processes, there is prospect of delaying their final development only by timely measures appropriate to their nature.

Thus the treatment of asthma aims at arresting the progress of the existing pathological changes, and at maintaining the healthy portions of the lungs in a state of greatest efficiency. These objects are quite attainable in most cases, partly by improving the nutrition of the organism and indirectly that of the lungs, partly by restoring the normal function of the bronchial surface.

I.—General Treatment.

1. *Hygienic Measures.*—The most reliable means of improving the general nutrition is undoubtedly the hygienic management of the disease. To derive from

measures of that kind all the benefit which they are capable of affording, they should be carried out systematically, and not left to the discretion of the patient. Asthmatics, as a rule, confident of the integrity of their organs, are only too apt to neglect the most obvious precautions for the maintenance of health, save, perhaps, those that refer to diet, because the transgression of these is immediately followed by the penalty; but even here the possession of some narcotic, by which the patients are able to relieve their "spasmodic breathing", often renders them incautious and callous. Hence the directions concerning hygienic measures should be at least as definite as those that are usually given with regard to the purely medicinal treatment.

a. Climate and Exercise.—Foremost among them is the constant supply of pure air. An atmosphere, containing more than 0·7 per cent. of carbonic acid, and the proportional amount of organic substances, is prejudicial to health. Such quantity of deleterious gases, however, accumulates only in confined places, and as asthmatics are, as a rule, of sedentary habits, they should, therefore, first direct their attention to an efficient ventilation of their dwellings. More especially is this needed in bedrooms at night, when oxygen should be stored up for consumption during the day. Not only the impairment of nutrition, but in many instances the nocturnal attacks of dyspnœa, are

due to an imperfect supply of pure air at night. The size of the bedroom affords not the least protection; however large it may be, the air in it is soon rendered insalubrious by the discharge into it of the products of respiration and perspiration. What is wanted, therefore, is the constant renewal of the air. But "the close windows", writes Sir James Clark,[1] "and thick curtains and hangings with which the beds are often so carefully surrounded, prevent the possibility of the air being renewed. The consequence is that we are breathing vitiated air during the greater part of the night, that is, during more than a third part of our lives; and thus the period of repose, which is necessary for the restoration of our mental and bodily vigour, becomes a source of disease. Sleep, under such circumstances, is often very disturbed, and always much less refreshing than when enjoyed in a well ventilated apartment; it often happens, indeed, that such repose, instead of being followed by renovated strength and activity, is succeeded by a degree of heaviness and languor, which are not overcome until the patient has been some time in a purer air.[2] Nor is this the only

[1] *The Sanative Influence of Climate.* 4th edition. London, 1864, p. 73.

[2] These are the cases of asthma in which coffee relieves the spasm. "It is often taken in the morning; and patients will tell you that, previous to taking their coffee, they are not fit for anything—can hardly move about; but that the taking of it is immediately followed by freedom of breathing, and an ability to enter at once on their daily occupation."—Hyde Salter, *op. cit.*, p. 202.

evil arising from sleeping in ill-ventilated apartments. When it is known that the blood undergoes most important changes in its circulation through the lungs, by means of the air which we breathe, and that these vital changes can only be effected by the respiration of pure air, it will be easily understood how the healthy functions of the lungs must be impeded by inhaling, for many successive hours, the vitiated air of bedrooms, and how the health may be as effectually destroyed by respiring impure air as by living on unwholesome and unnutritious food. . . . . Let a mother, who has been made anxious by the sickly look of her children, go from pure air into their bedroom in the morning, before a door or window has been opened, and remark the state of the atmosphere,—the close, oppressive, and often fœtid odour of the room,—and she may cease to wonder at the pale and sickly aspect of her children. Let her pay a similar visit, some time after means have been taken to secure a full supply and continual renewal of the air in the bedroom during the night, and she will be able to account for the more healthy appearance of her children, which is sure to be the consequence of supplying them with pure air to breathe."

Some patients may be readily induced to adopt the necessary means for the supply of pure air ; and the benefit which they invariably experience from it encourages them to continue the treatment. Others,

again, can in no way be persuaded to ventilate their rooms ; they confound ventilation and draught, and in their inordinate fear of "catching cold", they prefer, rather than risk this hypothetical danger, to expose themselves to the consequences inseparable from the inhalation of a poisonous atmosphere.

In dealing with this class of patients, it is well to remember that the effect of pure air upon those unaccustomed to it resembles almost that of alcohol. The chemical changes of the body, which hitherto have been at their lowest ebb, are by the free supply of that vital agent, at once roused to an energy previously unknown, and at times even so great as to produce an almost febrile condition. Moreover, if the patients have lived for a length of time in a confined atmosphere, their bronchial mucous membrane generally is in a state of anæsthesia from the narcotic influence of the carbonic acid ; but as, on exposure to a purer atmosphere, that membrane recovers part of its sensibility, cough is now readily induced by the irritating substances contained in the air. Such return of the bronchial sensibility, however, is usually regarded, not as a favourable sign, but as a "fresh cold". The patients, therefore, refuse to continue the treatment, and immediately return to narcotics, in order to suppress the cough. Hence, the longer they have breathed in a vitiated atmosphere, the more gradually are their habits to be changed ; otherwise, the neglect

of this precaution would deprive the sufferers of the benefit of the treatment, and cause the practitioner the annoyance of seeing his best efforts frustrated.

At all seasons of the year asthmatics should, if the weather at all permits, daily take exercise in the open air. There is no need for violent exertion, as often recommended, but the amount of the exercise should be apportioned to the requirements of each case. Gentle and sustained working of the muscles, as in walking, best answers the purpose ; it suffices to remove the waste products that accumulate in the body, and to effect a more complete oxygenation of the blood. The failing appetite is improved, and the impaired digestion and sanguification are restored ; so that exercise must be looked upon as the best of tonics. The difficulty only is, that the patients will not readily renounce their habitual indolence, either for want of energy on account of the imperfect decarbonisation of their tissues, or for fear of "catching cold". But the efforts, when once made, are always crowned with success ; even those in whom the disease of the lungs is considerably advanced, soon perceive the vivifying influence of oxygen, and a freedom of respiration to which they were strangers before. If active exercise is impossible, the passive should be substituted.

In the summer and autumn—from May till September—exercise in the open air is not always possible, on account of the dyspnœa which the pollen and

the spores of fungi floating in the atmosphere are likely to produce. So long, therefore, as the morbid sensibility of the bronchial mucous membrane persists, a sojourn at the sea-side is desirable. A place, however, which, though situated at the sea coast, partakes of the character of a bay, deeply indented into the mainland, is not so favourable for the purpose, especially if this bay is surrounded by land which is largely used for the growth of hay-grass.[1] But the more any sea-side place has the form and character of a small island, or of a narrow peninsula, and the wider the sea which surrounds either of these, the more completely will it protect the patient against the inconvenience arising from pollen in the air. For this reason a cruise in a yacht which can keep well out to sea is one of the best remedies that can be adopted, and, failing this, a sojourn on a small island in the open ocean is the best that can be found on land. Still, even at the sea-side, if the wind is blowing from the land, and if hay-grass is in flower at the time, the patient will be liable to have an attack of dyspnœa. It is, therefore, a matter of importance in selecting a retreat, to find one where the prevailing winds are from the sea, or one where the shore is backed with high cliffs, which act as a sort of screen when the land wind is blowing. Hyde Salter relates the case of a lady, who could breathe only in a cottage so near

[1] Dr. Blackley, *op. cit.*, p. 199-201.

to the sea, that in rough weather the waves broke against the walls of her house.[1]

Dr. Blackley[2] recommends, as places suitable for residence during the summer, Lundy Island, near to Ilfracombe, in the Bristol Channel; Lizard Point (Cornwall); the point of land near St. Mawe's, as well as many places of the south coast; some parts of the Isle of Man, and certain districts on the Welsh coast.

If the patient cannot take his residence at the sea-side, his best plan is to go to the centre of a large town—the larger and the more densely populated it is the better for him. These are the cases in which locality shows the caprice of asthma, and in which "the sooty air of London, Manchester, and Glasgow, has been found to be anti-spasmodic."[3] Perhaps the use of the respirator moistened with glycerine, or a wet sponge before the mouth and nose,[4]—with the view of filtering the inhaled air, may enable the patient to enjoy the advantages of out-door exercise, without exposing himself to the attendant inconvenience.

In the winter, asthmatics should reside at places where the genial climate and the beauty of the scenery both permit and attract them to spend the greater part of the day in the open air. Although other circumstances, such as the relaxation from many causes of

[1] *Op. cit.*, p. 285. [2] *Op. cit.*, p. 201.

[3] *Conf.*—Hyde Salter, p. 266-301.

[4] C. v. Nägeli, *Die niederen Pilze.* München, 1877, p. 154.

care and anxiety, the altered diet, etc., contribute to the benefit which is invariably experienced from residence at one of the numerous health resorts ; still, the main advantage of these is, that they afford opportunities for out-door exercise at a time when, were the patient in his own country, he would most probably be confined to his room.[1] In leaving home for the winter, the asthmatic should therefore distinctly realize the object of his migration to a different climate. He should remember that the atmosphere of the place which he seeks contains no special principle potent enough to penetrate into drawing and bed-rooms, and there to exert its curative influence.

It is agreeable to think that those advantages are not solely for those whom fortune has particularly favoured. Pure air sufficient for all the purposes of life are within the easy reach of even the poorest. "By keeping up the habit of going daily into the open air, in almost all weathers, under the protection of warm clothing, persons with very delicate lungs may bring themselves to bear this climate, and even strengthen their constitutions to an extent not generally believed."[2]

b. Gymnastics.—In feeble children, especially those whose thorax had been crippled by rickets, gentle gymnastics (Swedish) greatly assist the development of

[1] Sir James Clark, *op. cit.*, p. 10.

[2] *Ibid.*, p. 69.

the lungs. Athletic sports, if not carried to exhaustion, are still more useful. Adults also should bestow some attention upon the mechanism of respiration, in order to effect a complete ventilation of their lungs.

c. Baths.—The daily use of baths, in the summer, especially in the sea, is a very serviceable means of invigorating the constitution. Cold baths, however, are not suitable in cases in which the respiratory organs are unable to meet the increased demand for the production of heat consequent upon its abstraction. Tepid baths prove, under these circumstances, more beneficial, inasmuch as they greatly facilitate the performance of all functions. If the disease of the lungs is not far advanced, the advice of Dr. Chambers may be adopted.[1] He recommends, as an excellent device for invigorating the constitution, to take the morning baths by sitting in warm water, and having a bucket of cold water poured down the spine from the nape of the neck, and then being rubbed dry immediately with a rough-dried towel.

The systematic employment of warm baths of a temperature of from 95 to 102° F. has a direct curative influence upon asthma. By no other means than this is it possible so effectually to relieve the lungs of the chronic congestion with its abundant exudation of white flood corpuscles, which choke up the bronchi. The turgidity of the cutaneous surface

[1] *Diet in Health and Disease.* London, 1876, p. 281.

serves, as it were, to distribute the respiratory function between the lungs and the auxiliary organ—the skin. Carbonic acid is exhaled in larger quantities, the arterial pressure increases, and the discharge of the urine is augmented. Disease of the heart is not necessarily a contra-indication, but as the warm water tends to weaken the action of the cardiac muscles, stimulants may be required before the bath is taken. Niemeyer speaks highly of the good results which he obtained in inveterate cases of chronic cattarh ; he kept the patients for half-an-hour at a time in a bath of a temperature of at least 100° F., and then enveloped them in hot blankets, in which they remained for one or two hours longer. At first, as long as the dyspnœa was very severe, the patients suffered greatly, not only while in the bath, but during the sweating. Soon, however, generally towards the end of the first week, as the perspiration began to flow more readily and freely, they became satisfied at their improved condition, and were willing to continue the treatment. After eight or ten baths, the dyspnœa had abated in the most striking manner, and the cyanosis had disappeared.[1]

The addition of brine, or of Tidman's sea salt, from ten to fifteen pounds, improves the beneficial influence of warm baths, the temperature of which need not exceed 90° F. Brine baths are specially useful in

[1] *Text-Book of Practical Medicine.* 7th edition. London, 1870, p. 80.

scrofulous and rickety children. The famous sulphur baths of Barèges, Bagnères de Luchon, Cauterets and Eaux Bonnes, only act by the warm water, their contents of sulphuretted hydrogen being too small to produce an appreciable effect. The cures of "asthma" obtained at those places are greatly assisted by their climate. Lippert recommends vapour baths, if taken with the necessary precaution, in cases in which asthma depends upon a chronic congestion of the mucous membrane.[1]

d. Diet.—In regulating the diet of asthmatics, it is necessary to bear in mind that chronic gastric catarrh is as constant a symptom of their disease as are the dyspnœal seizures. Thus ere food can be expected to improve the general nutrition, the healthy function of the digestive organs should be restored as far as possible. The hygienic measures previously recommended, such as exercise in the open air, baths, etc., are valuable also in this respect; for, as the dyspepsia is due to the congestion of the gastric mucous membrane, consequent upon the obstruction of the pulmonary circulation, everything that tends to re-establish the freedom of the latter, favourably influences the power of the digestive organs for assimilation and absorption. Special treatment, however, is required for the removal of the abnormal fermentation, of constipation, and of flatulence; and such treatment is to be conducted

[1] Braun, *Balneotherapie.* Berlin, 1875, p. 638.

upon known principles, to which it is needless here to refer.

The quantity as well as the nature of the food should be such as to meet the requirements of each case. The age and constitution of the patient; the nature and duration of the disease; his appetite and habits; his tastes and idiosyncracies, and other like circumstances—all these should be considered in regulating the diet. A dietary table, applicable to all cases, is obviously impossible. It is by far the better plan to make each patient the subject of a special study, with a view of obtaining the necessary information as to his requirements. Errors of diet should be as much as possible avoided, if for no other reason at least for this, that they are most likely to provoke a dyspnœal attack.

As regards the quantity of the food, small and frequent meals should be the rule so long as the dyspepsia lasts. The interval between them depends on the time required not only for the assimilation of the different articles of diet, but also for their absorption; for, if ready-formed peptones are retained in the stomach, their presence interferes with the action of the gastric juice. The real wants of the organism are a safer guide than the craving of the appetite. The congestion of the gastric mucous membrane produces indefinite sensations which the patients erroneously interpret as promptings for food; and under the erroneous im-

pression that the more they consume, the better they are able to cope with their complaint, asthmatics are very prone to overload their stomachs. It is superfluous to say that only so much is beneficial as is assimilated and absorbed; the rest is not only useless, but even hurtful. Considering the paucity of the digestive juices secreted, and the impaired power of absorption—both the results of the venous congestion—the quantity of food should necessarily be small. A large meal of even the most digestible articles is sure to disagree with the patient, and to provoke an asthmatic seizure. No food should be taken shortly before going to bed; but time should be allowed for the completion of the digestive process before the patient retires to rest.

The quality of the food is of less importance than its quantity. There are no articles absolutely digestible or indigestible; but what is easy of digestion for one asthmatic, may not be so for another. The digestibility of food depends on its chemical composition, its shape, its preparation, etc. Upon the whole, the diet of asthmatics should, as in the healthy, contain the necessary proportion of albuminous and hydrocarbonaceous substances. In the young and feeble patients, hydrocarbons and especially fat should slightly preponderate; in the obese, in whom a reduction of the corpulence is desirable, a suitable regimen should be instituted. It is a mistake, however, to cut down

diet to a rigid monotony, and erase from the future bill of fare everything that has once seemed to be followed by indigestion. On the contrary, the more hopeful course is to add to the dietary everything that has been once found to agree. Thus a wholesome and extensive choice of articles will be obtained.

Some articles are looked upon as highly "asthmatic", and should therefore be avoided. They are generally those that readily undergo abnormal fermentation, or are decomposed into carbonic acid and hydrogen, as malt liquors, sugar, preserved fruits and cheese. During meals, the attention of emphysematous patients especially should not be diverted from the act of swallowing, since by inadvertence, particles of food may easily enter the air passages.

2. *Treatment of constitutional anomalies and local affections.*— The causal indication demands an appropriate treatment of the constitutional anomalies and local affections with which asthma is generally connected. The appearance of dyspnœal paroxysms in a child, in whom the presence of rickets may be suspected, calls for a suitable dietetic regimen and for the exhibition of lime water. Benecke warmly recommends in those cases the hypophosphite of lime, which he considers as a means of "manuring the infantile organism."[1] That salt however is insoluble in the intestinal

[1] Hermann Köhler, *Handb. d. physiol. Therapeutik.* Göttingen, 1876, p. 135.

canal, and is not absorbed The phosphorus needed for the formation of bone is amply provided by the oxidation of the protein substances. Hermann Köhler, by no means sceptical as regards the efficacy of drugs, is of opinion that hypophosphite of lime is, "without a proper respiratory diet", useless, and with it, only of doubtful utility.[1] At a later period, when the rickety deformities have developed themselves, as also in cases of scrofulosis, the exhibition of iron, and particularly of cod-liver oil, serves not only to improve the general nutrition, but also to arrest the progress of the morbid changes in the lungs.

Hydrotherapy may be cautiously tried, where a scrofulous diathesis exists. Rilliet and Barthez have obtained good results from it in a case in which asthma was produced by enlargement of the bronchial glands.

In patients with a tendency to corpulence, and also in the gouty, the impediments of the respiration are best avoided by the regulation of the diet, by exercise and by the use of Carlsbad or Vichy water. A season at these places is often productive of great benefit. In obese females, sterile or past child-bearing, the disposition to asthma is often considerably relieved by repeated incisions of the os uteri if, for some reason or other, attention is directed to the generative organs. Doubtless, both the dyspnœa and the

uterine disturbances are the consequences of a general plethora; and though the prevalent theory ascribes the former to a nervous derangement produced by the latter, that theory may be allowed to pass, so long as its practical application benefits the patient. The same favourable modification of asthma is observed in plethoric individuals after a hæmorrhoidal flux. But in either case, the benefit is only transient, as the patients strive by all possible means to repair the loss of blood. At the present time, when a remedial measure of great usefulness—the lost art in surgery—has been quite discarded, because it had formerly been abused, one does not dare to recommend the adoption of a practice, founded though it may be upon clinical experience.

If asthma is connected with diseases of the heart, iron and digitalis serve to strengthen and to regulate the cardiac action. The cases most difficult to deal with are those in which the dyspnœa is due to the so-called brown induration of the lungs, in consequence of a congenital hypoplasia of the arterial system. All the resources of art often fail to prevent the constant emigration of white blood corpuscles which obstruct the bronchi.

If asthma complicates renal affections, the patient should for a time be kept in bed. Gentle but persistent diaphoresis effects as much as may be expected under such circumstances.

a. Medicinal.—Among the numerous remedies that have been recommended in the treatment of asthma, the great majority are narcotics or so-called nervine tonics,[1] intended to allay the supposed morbid irritability of the centre of respiration. But apart from the uncertainty of their operation, even in the hands of those who most loudly proclaim their virtues, their prolonged exhibition is neither indicated by the nature of the disease, nor has experience shewn them to be productive of more than a transient benefit.

The medicinal agents which in suitable cases appear to be useful are :—

1. *Arsenic.*—The employment of this drug in asthma is mainly founded on the observations of its effect upon the habitual arsenic eaters in Lower Austria and Styria. According to the concurrent testimony of many trustworthy informants, there are in those countries men, of the lower classes, who resort to arsenic for the alleged purpose of rendering themselves, or rather their respiration, more apt for ascending mountains, and for hunting. They generally commence at the age of eighteen, by taking small doses of arsenic, and gradually attain such tolerance of it, as

[1] Chloride of platinum (Huss), zinc, silver (Kopp), in R. Köhler's *Specielle Therapie*, 1868. Bd. i, p. 656 ;—Antonio Curci, "Azione del argento sul sistema nervoso e moscolare".—*Lo Sperimentale.* Dec. 1875.

to be able to consume from four to six grains once or several times a week. Under the influence of that drug they indeed endure great physical exertion without embarrassment of their breathing; their appetite and digestion are good, and their nervous system is in a state of great vivacity. They, moreover, appear to be singularly free from disease, and, as a rule, reach good old age; but the omission of the arsenic is always followed by indisposition, so that they are forced to return to its use as soon as possible.

The physiological action of arsenic is as yet too imperfectly known to admit a satisfactory explanation of the facts just mentioned. That it enters the circulation is evident from its appearance in the bile, in the saliva, in the perspiration, and in the urine; but what changes it produces in the blood, and how it affects the nervous centres are, for the present, mere matters of speculation. But it is doubtful whether the good results obtained by its use, in the case of those mountaineers, are due to its remote effects. Arsenic is known to be a useful stomachic,[1] and as the men who resort to it are in the habit of consuming large quantities of ardent spirits, they apparently possess in it an agent, which, by stimulating their appetite, and assisting their digestion, preserves their nutrition, and enables them to follow their employment, notwithstanding their intemperance, which would

otherwise speedily incapacitate them. But their healthy occupation is by no means a subordinate factor in the improvement of the general nutrition, and there is no proof that arsenic produces precisely the same effect in those who do not pursue the same mode of life. Indeed, the conflicting opinions which physiologists arrived at concerning the increase or reduction in the excretion of carbonic acid and of urea, and concerning the gain or loss of the weight of the body, seem to be attributable to the differences of external circumstances under which their experiments were conducted.

Thus in these, and doubtless in other cases, arsenic in medicinal doses owes its virtue to its properties of —1, arresting fermentation[1]; 2, stimulating the appetite and assisting digestion; and 3, improving the general nutrition. The irritation which it produces in the gastric mucous membrane gives rise to a pleasant sensation of warmth in the stomach, and to a feeling akin to hunger; and as its presence in no way interferes with the action of the saliva, nor with that of the gastric and pancreatic juices, the food consumed in larger quantities is also completely digested. There is, moreover, according to Vaudrey,[2] a copious evacuation of pultaceous stools—a circumstance that probably accounts for the freedom of respiration generally experienced.

[1] Savitsch and Nicolai Johannson, quoted by Köhler, *loc. cit.*, p. 725.

[2] *Ibid.*, p. 716.

On the erroneous supposition that arsenic exerts a specific influence on the respiratory organ, Trousseau revived the practice recommended by Ettmüller, of inhaling the drug for the relief of asthma.[1] Buchheim denounces this procedure as dangerous.[2] Trousseau himself is silent on the results which he obtained, but Waldenburg[3] states that the inhalation of arsenic is much less efficacious than its internal administration. Viand Grand-Marais[4] and Fred. Julius,[5] on the contrary, claim advantages from arsenical fumigations; but it appears that the mere irritation which such fumigation produces upon the respiratory mucous membrane facilitates expectoration in cases of chronic bronchitis—an effect more readily attainable by less dangerous means.

Arsenic is thus indicated for the relief of the dyspepsia of asthmatics[6] and for the improvement of their nutrition, provided the alimentary canal is free from inflammatory affections and the action of the heart is not too feeble. It is best given as Fowler's solution, or Liqu. Sod. Arsen., immediately before meals; corrosion of the gastric mucous membrane need not be feared, as arsenic possesses no affinity to the albumen

[1] Hirtz, Art. "Arsenic".—*Nouveau Diction. de Médecine*, etc., tome iii, p. 119.

[2] Lehrbuch der Arzneimittelehre, 1876, p. 311.

[3] *Lehrbuch der respiratorischen Therapie.* Berlin, 1872, p. 488.

[4] *Moniteur des Hôpit.*, Juin 28-30, 1850.

[5] *Lancet*, vol. ii, August 1861.

[6] Anstie, quoted by Köhler, *op. cit.*, 736.

of the tissues. The treatment is best begun with the exhibition of a laxative, in order to remove the fæcal matter from the intestinal canal. The dose is gradually increased so as to reach its maximum within fourteen days; it may then be continued for some time, and subsequently gradually diminished. On the appearance of pain in the epigastrium, the drug should be at once omitted. The patient, moreover, should be carefully watched, as even the smallest doses have the tendency to weaken the energy of the heart.[1]

It is needless to add that the "herpetic eruption" which forms an integral symptom of hay asthma does not require the exhibition of arsenic.

In cases in which asthma is due to compression of the air tubes by enlargement of the bronchial glands, arsenic may be cautiously tried. Commencing with five drops of Fowler's solution, Billroth[2] increases the dose in periods of two or three days to ten, fifteen, twenty-four, and even forty drops, and again diminishes it in similar proportion. If signs of poisoning appear, the treatment should be interrupted. After the fourth or fifth day of treatment, the temperature generally rises in the evening (101·3°—103·1° Fahr.), but becomes normal in the morning; and this febrile state, which Harless also had noticed,[3] lasts even for a couple of days after the

treatment has been discontinued. But it is by no means desirable to carry the effect of the drug so far.

2. *Alkalies.* — In the cases in which viscid mucus occludes a number of bronchioles, so as to impede the access of air to the lungs, the means previously mentioned as intended to reduce the disposition to asthma, remain ineffectual in consequence of the imperfect oxygenation of the blood. Under these circumstances, alkalies may be usefully given, with the view of removing the existing obstacle of respiration and of modifying the secretory function of the bronchial mucous membrane.

Since the time of Laennec, alkalies have been employed for the purpose of "dissolving" tenacious sputa, and there can be no doubt that they possess this property in an eminent degree. On account of their great diffusibility, they are rapidly absorbed into the blood, the plasticity of which they diminish ; but as the blood strives to maintain its fixed standard of alkalinity, they are rapidly eliminated and reappear in the various secretions. In what manner they alter the exosmotic equivalent of the blood is not quite known ; but certain it is that their carbonates and chlorides favour the formation of mucus, which, according to Nasse[1], contains seventy per cent. of chloride of sodium.

As in the present case the alkalies have to be continued for a length of time, the salts of soda are preferable be-

[1] Buchheim, *op. cit.*, p. 108.

cause they are better borne by the stomach than those of potash. They are best given in the shape of a mineral water, such as Ems, Wiesbaden, Kissingen, Vichy, etc.

Iodide of potassium, which forms the base of Aubrée's well-known specific, was first given in asthma by Horace Green. It has all the properties of an alkali, and possesses moreover, as Heubel[1] supposes, a special affinity to the pulmonary tissue. Its administration in suitable cases is always attended by the best results.[2] The dose varies from five to ten grains several times a day. It should be freely diluted, and be taken upon an empty stomach. In the doses just mentioned it does not spoil, but rather improves appetite and digestion.

Iodide of potassium is inapplicable, however, when the bronchial mucous membrane is in a state of acute inflammation. For, in contact with the atmosphere, the iodine is disengaged from its combination with the albumen of the blood, and when thus set free, it is apt to irritate the surface of the air tubes, and to produce here an analogous process, as it does upon the skin and the nasal mucous membrane.

Germain Sée[3] recommends bromide of potassium, and reports three cases in which its exhibition proved suc-

[1] H. Köhler, *op. cit.*, p. 540.

[2] Hyde Salter, *op. cit.*, p. 302-8; James Lawrie, "The Therapeutic Value of Iodide of Potassium". *British Med. Journ.*, Jan., 1875; Stabell, "Asthma nervosum", Virchow's *Jahresb.*, 1875, Bd. ii., p. 207.

cessful. This salt, however, acts chiefly through its halogen, which diminishes the reflex irritability of the nervous system, but it is less efficacious for the purpose here required.

Chlorate of potash also may be advantageously given. It appears to be for various reasons even preferable to iodide of potassium. Isambert found from ninety-five to ninety-nine per cent. of the salt in the secretions.[1]

The remedies that occupy but a doubtful place in the treatment of the asthmatic tendency are :—

3. *Balsams.*—Turpentine and assafœtida have been recommended, the former by Trousseau,[2] and the latter by Pereira[3] and Marshall Hall.[4] Like all remedies of this class, they are eliminated through the bronchial mucous membrane, and stimulate in their passage the mucous glands. The secretion is thus rendered more liquid, and is therefore less liable to induce dyspnœa. Yet, notwithstanding this beneficial effect upon the respiratory surface, they produce, if continued for some time, nausea, vomiting, colic, and diarrhœa ; so that in the weakened state of the digestive organs of asthmatics, such disturbances ought to be avoided. It is preferable, therefore, if their specific action is required, to employ them in inhalations.

Grindelia Robusta (Nuttall), has been recommended by some American physicians as a useful antispasmodic remedy in asthma.[1] It is a plant of the composite family, a native of California, and contains, so far as the scanty information on the subject goes, an oleo-resin. An infusion and also an extract are prepared of the flower-heads, to both of which borax is added. The extract is given in doses of from three to six grains three times a day.

4. *Sulphur.*—Sulphur, once a famous expectorant, has been given by Duclos for the purpose of removing the "herpetic diathesis", which he regards as the cause of asthma. Trousseau, who has tried the remedy, evidently does not share the illusions of Duclos as to its "prodigious power". The benefit which asthmatics occasionally obtain from treatment at the Pyrenees baths is due much more to the climate of those places and to the warm water than to the sulphur of their springs. Even the richest amongst these, Bagnères, contains only one grain of that ingredient in sixteen ounces of water, of which the dose varies from one tablespoonful to three ounces twice a day.[2]

β. Mechanical.—The mechanical treatment of the respiratory tract greatly assists, so far as it is prac-

[1] Henry Gibbons, *Americ. Journ. of Medic. Science*, April 1867, p. 565; Alabneda Gibbons, *Philad. Med. and Surg. Report*, September 19th, 1874, p. 225.

[2] Pidoux, *Journal de Thérapeutique*, No. 2, 1874; Virchow's *Jahresbericht*, 1874, Bd. i, p. 554.

ticable, the various constitutional remedies. Its effective operation, however, is almost entirely limited to :—

1. *The mucous membrane of the nose.*—This part of the air passages demands a larger share of attention than it has hitherto received in the management of asthma ; for there is little doubt that the permeability of the nasal canal is necessary for the due performance of the respiratory function, and for the protection of the respiratory surface. The buccal cavity cannot adequately replace the "true respiratory duct".[1] If the latter is obstructed, the supply of air to the lungs is considerably diminished, as may be readily ascertained on listening to the chest of one who alternately breathes through the mouth and the nose. In infants, occlusion of the nose may prove fatal, and there are cases in which, even in adults, paralysis of the nasal muscles has been known to give rise to extreme dyspnœa, which was promptly relieved, however, by dilating the nostrils.[2] But the nose is, as previously mentioned, an important filtering apparatus, inasmuch as the foreign particles floating in the air are arrested on its lubricated vibrissæ, and are thereby prevented from penetrating to the lungs. Milne Edwards, moreover, attributes to it the function of adjusting the temperature of the inhaled air to the

[1] Bérard, *Cours de Physiologie*, 56e Leçon, p. 286.

[2] Traube, "Klin. Mittheilungen".—*Berl. Klin. Wochens.*, 1871, No. 25.

requirements of the bronchial surface, and of providing for its necessary degree of moisture.[1] Thus, in the cases observed by Voltolini[2] and Haenish,[3] the removal of nasal polypi served to reduce the disposition to asthmatic seizures; and, it is needless to add, that, where such causes of obstruction exist, the necessary operation should not, if possible, be delayed. Generally, however, the occlusion of the canal is due to a chronic congestion of the mucous membrane, which as a rule is greatly diminished by the measures that tend to restore the freedom of the pulmonary circulation. Yet, not infrequently, scabs, mucus, and foreign bodies accumulate within the nasal canal, and maintain a state of inflammatory process which can be effectually relieved only by means of the nasal douche. Tepid water, with the addition of a small quantity of chloride of sodium, best answers the purpose, and there need be no fear that, with proper precautions, any of the fluid enters the Eustachian tubes.[4] Astringents are seldom required, and if so, cold water or a weak solution of sulphate of zinc (one half

[1] *Anatom. et Physiol. Comp.*, t. ii, p. 266.

[2] *Op. cit.*, p. 249, *seq.*

[3] "Zur Ætiologie u. Therapie der Asthma Bronchiale."—*Berlin. Klin. Wochensch.*, 1874, p. 503.

[4] Martin F. Coomes, "Nasal Catarrh and its Treatment".—*Philad. Med. and Surg. Report*, Dec. 4th, 1875. Paul, "Note sur l'irrigation nasale", etc.—*Bulletin génér. de Thérapeutique*, Août 30, 1875.

grain to the ounce), appears to be most serviceable. In order to remove the susceptibility to foreign bodies, particularly pollen grains, calomel or the yellow oxide of mercury, in the shape of snuff-powder or ointment, may be advantageously employed.

The application of caustic ammonia to the posterior nares as recommended by Ducros, and modified by Trousseau, is a dangerous practice.[1] Its efficacy consists in that, as Buchheim suggests, small quantities of ammonia enter the air passages and stimulate expectoration — a supposition corroborated by the fact that in one of Trousseau's cases, such application was followed by œdema of the glottis. If the topical effects of alkalis are desired there are safer means of obtaining them.

2. *Bronchi.*—Although the invention of the spray-producing apparatus has brought the surface of the air tubes also within the reach of local treatment, experience has not confirmed all the hopes founded on that practice. Probably the fault rests not so much with the method itself, as with the nature of the disease which it is intended to relieve; for there are few, if any, cases of chronic bronchitis, in which the morbid changes are limited to the internal layer of the bronchi, whereas upon their deeper structures inhalations have no appreciable influence. Still there are numerous instances, in which inhala-

tions, continued for some time, do prove useful, and Waldenburg records some, in which such treatment is said to have *completely* cured the disposition to asthma.[1]

However this may be, the deep inspirations which patients are directed to take whilst inhaling—the gymnastics of the lungs[2]—are, no doubt, most beneficial to those who do everything for the cure of their dyspnœa but breathe properly, and whose nervous system, moreover, is blunted by a long use of narcotics. Not the less efficacious is the watery vapour that is drawn into the air-passages, and bathes, as it were, the diseased surface of the bronchi.

The medicinal substances which Waldenburg has found most useful for inhalations are: chloride of sodium or of ammonium, together with oil of turpentine. The former is intended to soften the viscid mucus, the latter to stimulate the mucous glands. About one ounce of the solution is inhaled at the time, containing from two to fifteen grains of the alkali, and from two to twenty minims of the turpentine.

3. *Respiratory Surface.*—Inhalation of compressed air, in chambers especially prepared for the purpose, is greatly appreciated by the patients who have tried it. Physicians, who, as proprietors of such chambers, have occasion to observe the effect of that treatment, are, although unanimous as to its good results, yet constrained to admit that those inhalations often fail to relieve the

[1] Waldenburg, *op. cit.*, pp. 483 & 488. [2] *Ibid.*, p. 481.

"purely nervous asthma", while in the complicated forms of the disease they merely render the dyspnœal seizures less frequent and less severe.[1] As to the manner in which condensed air acts, nothing is definitely known. The theory that under the greater pressure a larger quantity of oxygen is absorbed is, upon physiological grounds, contested by Buchheim[2] and Rohden.[3] The latter maintains that, though under the greater pressure oxygen may be temporarily diffused in the serum, yet this surplus of the gas escapes when the pressure has ceased.

The views of Buchheim and Rohden are based on the experiments of Seguin and Lavoisier,[4] and on those of Regnault and Reiset.[5] Animals in an atmosphere of pure oxygen do not consume of this more than in the ordinary air. Pflüger,[6] moreover, has shewn that in breathing the ordinary atmosphere, the arterial blood is approximately saturated with oxygen, and that, however abundant the supply, its absorption is not proportionately increased. The observations of Voit

[1] I. Lange and Bertin, *Conf.*, R. Köhler, *op. cit.*, p. 656; Knauth, *Handb. d. pneumatischen Therapie.* Leipzig, 1876, p. 73.

[2] "Ueber die therapeutische Verwendung des Sauerstoffs."—*Arch. f. d. Experim. Pathol. u. Pharm.*, Bd. iv, 1875, p. 142.

[3] Braun, *op. cit.*, p. 641.

[4] *Mémoires de l'Académie des Sciences*, 1789, p. 573.

[5] *Récherches chimiques sur la respir.*, etc. Paris, 1849.

[6] "Ueb. d. Ursache der Athembeweg."—*Arch. f. d. ges. Physiol.*, Bd. i, 1868, p. 69.

and Pettenkofer[1] in man led to similar conclusions, and there can be no doubt that the affinity of the hæmoglobin to oxygen is definite, and satisfied by a relatively small quantity.

Demarquay and Leconte,[2] on the contrary, allege that dogs, on inhaling pure oxygen, become more lively and more voracious than usual. The results of these experiments, however, require confirmation. For the present, there is as little ground for assuming that a supply of oxygen greater than that contained in the atmospheric air, improves respiration, as there is that overloading of the stomach improves nutrition.

The efficacy of those apparatuses can therefore be attributed only to the compression of the intestinal gases, which permits the diaphragm a greater freedom of action. Paul Bert was informed by the men who worked under a greater atmospheric pressure, that they were always forced to tighten the belts round their waists on account of the retraction of the abdomen.[3] Whether, as Knauthe thinks, the gradual increase and the gradual decrease of the pressure on entering and leaving the air-chamber restores the deficient elasticity of the lungs,[4] is as yet mere matter of opinion. However this may be, the

[1] Quoted by Gorup-Besanez.—*Lehrb. d. Physiol. Chem.*, 1867, p. 725.

[2] *Comptes Rendus*, 1864; quoted by Gorup-Besanez, *op. cit.*, p. 721.

[3] *Pressions Barométriques*. Paris, 1878, p. 517.

question is as yet of too little practical importance ; for, besides being costly, and within reach of but a few, the remedy has avowedly only a palliative effect.

The inhalation of, or rather the expiration into, rarified air, in cases of emphysema, appears to be more practicable on account of the greater simplicity of the instruments employed for the purpose. Starting from the supposition that the dyspnœa accompanying that affection was due to the loss of the pulmonary elasticity, Hauke[1] contrived an apparatus intended to supply, by a *vis a fronte*, the deficient *vis a tergo*. A trial with that instrument afforded, to all appearances, well-grounded hopes as to its usefulness. Its unwieldiness, however, induced me to suggest some modifications,[2] with the view of insuring the widest possible application of a treatment that seemed so eminently rational,—not indeed for the cure of the disease, but for the relief of its most prominent symptoms. But a larger experience has, subsequently, convinced me that even the most extensive emphysema is, as such, not productive of dyspnœa, but that this, if present, is always due either to hyperæmia, or to inflammation of the mucous membrane, or to obstruction of the bronchi. Under these circumstances I cannot but hesitate to attribute the relief, which I observed in several cases, to the use of the apparatus. Still, the principle upon which that method of treatment is based is

[1] Ein Apparat für künstliche Respiration, etc., Wien, 1870.

[2] *Lancet*, Nov. 25, 1871.

so generally approved of, that there are at present fourteen different kinds of such instruments in more or less extensive operation. One of these, devised by Waldenburg, is said to possess many advantages over all the others. The success that Waldenburg obtained by it is indeed very striking. In one patient, aged seventy-four, suffering from senile emphysema, the over-distension of the lungs completely disappeared within ten days[1]; whereas in the younger patients subject to asthma, bronchitis, and emphysema, a longer treatment was required, which invariably resulted in the removal of all the subjective and objective symptoms. It is difficult, however, to assign to the expiration into rarified air its true share in the cure of those cases; for, besides the instrument, Waldenburg employed also inhalations of chloride of sodium and of turpentine, together with the internal administration of arsenic—means which by themselves[2] have in the same diseases been equally effective in his hands.

c. *Treatment of the Paroxysm.*

Attention to the obvious rules of hygiene affords the best protection against the recurrence of the dyspnœal paroxysms. Any infraction of those rules is promptly followed by its penalty, and that experience would be the safest guide of asthmatics, were it not that

[1] *Die pneumatische Behandlung*, etc. Berlin, 1875, p. 410.

[2] *Comp. Pneumat. Behandl.*, etc., p. 381-410; and *Respir. Therapie.*, p. 487-508.

they are too much given to rely on their inferences rather than on facts, and thus by endeavouring to avoid Scylla, they generally drift into Charybdis. It is well, therefore, to acquaint them with the nature of the usual provocatives of the attacks, and to impress upon them that the return of these rests, in a great measure, with themselves. A too rigid prophylaxis, however, is impracticable, and too stringent directions, with a view of avoiding all possible causes of asthma, are sure to be neglected. Timid and nervous patients would, in this way, only be kept in a state of continual anxiety of an impending dyspnœa—an anxiety that vitiates all relish of life; and the more daring among them would risk an occasional attack rather than submit to numerous restrictions. Moreover, there are circumstances over which patients have not the least control, and thus the safest means of escaping their injurious consequences is to diminish, by rational treatment, the susceptibility to them.

The treatment of the paroxysm necessarily varies according to the nature of each case, so that an accurate diagnosis is the first condition of success. The prodromal symptoms, where such exist, afford valuable indications, by acting upon which the attack may either be prevented, or promptly relieved. Thus there are on record numerous instances in which, upon the appearance of the signs peculiar to carbonic acid intoxication, as headache, vertigo, fretfulness of temper,

and pain in the muscles, an efficient ventilation of the lungs—by walking, driving, riding on horseback, etc. —successfully averted the threatening, and completely cured the fully developed, dyspnœa. Frequently, however, the precursory phenomena are either wanting or not so conspicuous. In that case, if the diagnosis remain doubtful, the treatment is necessarily deprived of its reliable basis.

1. *Causal Indications.*—Asthmatics, as a rule, adopt of their own accord such measures as are best calculated to assist them in their respiratory efforts. They assume a posture most convenient to themselves; they remove articles of clothing that impede their breathing; and they open the windows to obtain a supply of fresh air. It is but rare that special directions as to these points are needed. But it is by no means a good plan to advise indiscriminately every patient to rest, on the approach of an attack, his elbows on a table,[1] and to wait with resignation for the cessation of the "spasm". An obstruction of the bronchial tubes is often more readily removed by some muscular exertion than by deliberate respiratory acts.[2]

a. Irritating Gases.—The measures just alluded to suffice to restore the freedom of respiration in those

[1] Hyde Salter, *op. cit.*, p. 183.

[2] Conf. *Cases of Emotional Asth.*, Hyde Salter, *op. cit.* p. 210,

comparatively mild attacks in which the dyspnœa arises from the inhalation of irritating gases. There are no remedial agents capable of chasing from the tissues and from the blood the carbonic acid that accumulates in them; and the hyperæmia of the bronchial mucous membrane requires no special treatment, since, with the removal of the exciting cause, it tends of itself to recovery. Such expectation will not appear inopportune, considering that, in cases of this kind, the happiest results are often obtained[1] "by the most blind and purposeless treatment". Hot and strong coffee, however, proves occasionally very useful when the energy of the heart is temporarily weakened by an excess of carbonic acid in the blood.

b. Flatulence.—The dyspnœa produced by a loaded condition of the stomach or of the rectum is speedily relieved by an emetic or an enema. If the offending substances, however, cannot be at once removed by either of these means, there is often a flatulent distension of the intestines that resists for a time all possible treatment. Preparations of ammonia and carminatives have no marked effect; ice, as recommended by Romberg, is much more beneficial; but the best results are then obtained by small doses of creosote in camphor or cinnamon water.

c. Dust.—The fluxionary hyperæmia or inflamma-

[1] Hyde Salter, *op. cit.*, p. 104.

tion of the bronchi, that arises from the inhalation of dust, can be subdued only by the removal of the irritants. This, however, is often a matter of great difficulty, especially if the foreign bodies firmly adhere to the mucous membrane. The dyspnœa may thus last for days, or even longer, until the particles of dust are detached from the bronchial surface by a copious exudation of serum.[1] In the cases in which asthma is due to the action of pollen grains, inhalations of emollients, or of warm water, bring generally some relief.[2] Such inhalations, however, are, in consequence of the dyspnœa, but imperfectly performed, and even at the best, the quantity of watery vapour that thus enters the air tubes is far too small to produce a decided effect. What is wanted is a rinsing, as it were, of the bronchial surface. Alkalies, although they increase the bronchial secretion, are yet too slow in their action. Copious draughts of warm water would best answer the purpose; but it is preferable to give hot infusions of Flores Sambuci or of Senega root, to which acetate or carbonate of ammonia may be added.[3]

[1] No doubt the diaphoretic drinks, as hot grog, or such medicines as Dover's Powder, owe their reputation of curing "colds" to this, that by the copious transudation which they produce, they cleanse, as it were, the bronchial lining of its irritating substances.

[2] Langenbeck and F. W. Mackenzie, in *Phöbus*, *op. cit.*, pp. 207 and 237.

[3] Ernest Meyer and Gordon, in *Phöbus*, *op. cit.*, pp. 216 & 222.

In this form of asthma, alcoholic stimulants are superfluous. The relief that has been known to follow their exhibition, is really due to the warm water in which they are given. The experience of the patients is conclusive on that point. One patient emphatically declares that "if she took the gin with cold water, she might take any quantity and it would do her no good, for if she lets it stand till it is cool, and then takes it, it is useless".[1] Another who, on account of his dyspnœa, consumed twelve gallons of brandy in two months, was not the less convinced that the warm water was really the effective remedy, and he even insisted "that the water should be boiling".[2] Alcoholic stimulants should not be prescribed, for, as asthmatics return to them whenever their dyspnœa appears, they contract, by gradually increasing the dose, habits of intemperance, from which they can never be weaned.

Jaborandi has been recommended by Albert Robin and Gubler,[3] and in the cases which they observed the dyspnœal seizures completely subsided within one hour, while the expectoration became more loose, and the sonorous rhonchi disappeared. These results are quite in accordance with the physiological action of the plant; for in the majority of instances the exhalation from the lungs increases *pari passu* with that

[1] Hyde Salter, *op. cit.*, p. 205.

[2] *Ibid.*, p. 207.

[3] Virchow's *Jahresbericht*, 1875. Bd. i., p. 521.

from the skin. Jaborandi deserves, therefore, a trial in asthma produced by the inhalation of foreign bodies. It is difficult, however, to persuade a patient to take the drug a second time, on account of the very unpleasant effects, such as nausea, vomiting, salivation, and strangury, which accompany its administration.

Ipecacuanha, tartar emetic, and lobelia, in large and nauseating doses, have frequently been found useful. Their efficacy is due to their power of reducing the arterial pressure, so that by the consequent increase of the serous exudation, foreign bodies are more easily detached from the bronchial mucous membrane.

The smoking of tobacco and of stramonium acts in precisely the same way, the difference being only one of degree. Tobacco, however, is much less used in the treatment of asthma than stramonium, the virtues of which are greatly extolled by advertisements in the daily papers. This drug, which forms the principal ingredient of various kinds of cigarettes, owes, no doubt, the reputation which it enjoys much less to the deliberate judgment of physicians than to the force of public opinion. Travelling gipsies are said to have, during the middle ages, imported into Europe the practice of smoking stramonium—a practice that, according to Sauvage and Boerhaave, had been utilised for criminal purposes. Formerly, stramonium seems to have been employed, on account of the pleasant hallucinations

which it produces, in the treatment of melancholia.[1] But its more general application in asthma dates from the time when Mr. Sills, a merchant residing at Hackney, sent to the daily papers a report of the benefit which he had obtained by it in his own case. Physicians apparently discountenanced the smoking of stramonium; for, in order to show that their prejudice was unfounded, Mr. English, a surgeon of Great Tower Street, related how his own asthma had been relieved by it.[2] Mr. English suffered for a long time from bronchitis with fibrinous sputa. "In the mornings," he writes, "what is expectorated is often streaked with blood, and sometimes a little pure blood is coughed up.[3] The first time I smoked, I expectorated from the bronchia pieces of clear congealed phlegm, from one-half of an inch to about one inch in length, and the thickness of a crow's quill."[4] But he significantly adds, "When I commenced smoking the stramonium the paroxysm was declining."[5] The experiments of Dr. C. J. B. Williams supplied a scientific basis for the employment of that drug; for, on poisoning a dog with thirty grains of extract of stramonium, no trace of contractility could be detected in the lungs.[6] Subsequent experiments have fully confirmed the fact that daturin, which, indeed,

[1] Christie, *Edinb. Med. and Surg. Journ.*, vol. vii, 1816, p. 158.
[2] *Ibid.*, p. 277, *seq.*

is identical with atropin, reduces and even annihilates the contractility of the unstriped muscles; but it is deserving of notice that an imperfect decarbonisation of the blood—one that doubtless exists during the asthmatic seizure—antagonises the effect of stramonium, and restores the muscular contractility.[1] However this may be, in smoking stramonium, the effect of daturin is quite out of the question, because no such substance exists. The products of dry distillation, *i.e.*, smoking of stramonium and of tobacco, are not daturin and nicotin, but the members of the picolin series, as picolin, pyridin, lutidin, and collidin[2]; and the same substances are obtained from the combustion of such innocent materials as, for instance, taraxacum and willow.[3] Picolin and pyridin irritate, if inhaled, the bronchial mucous membrane, which is found in a state of great congestion accompanied by œdema;[4] while their internal administration, as it occurs by swallowing the saliva in smoking, gives rise to congestion of the lungs and of the nervous centres—a congestion often so intense as to lead to extravasation of blood.

The physiological action of tobacco and stramonium renders them, indeed, suitable in the asthmatic dysp-

[1] Hermann Köhler, *op. cit.*, p. 1005.

[2] Vohl u. Eulenberg, "Ueber Tabak in *Toxilogischer Beziehung*". *Vierteljahrsch. f. gericht. u. offent. Mediz.* Neue Forge. Bd. xiv, 1872, p. 254. [3] *Ibid.*, p. 281. [4] *Ibid.*, p. 206.

nœa, for the purpose of meeting the causal indication. But apart from the dangerous state of collapse which they are apt to produce, there is no one physician who has seen any decided benefit from them. Christie mentioned that "in none of these cases did it ever fail to relieve, when used *before the accession of the paroxysm*, or even after the commencement, but I am sorry to say that it did not often prevent the repetition of the fit, unless great attention was at the same time paid to diet and regimen".[1] Bree tried it in eighty-two cases ; in fifty-eight of these it had no permanent effect, and, in the remaining twenty-four it acted injuriously.[2] Hyde Salter is only lukewarm in its favour, and his experience is that, in the majority of cases, it gives *some* relief. But he thinks it is more effective in the way of prevention than cure ;[3] he gives the extract internally, and advises smoking of a pipe of the herb at night whether an attack of asthma be threatening or not. In this way the drug acts as an expectorant, and, as such, it is hardly the fittest that can be selected. But Hyde Salter correctly points out that, during a severe fit, the patient has not breath enough to smoke, and, in those attacks in which he is able to do so, it is no excess of scepticism to say that the remedy is certainly of very subordinate value. In hay-asthma, tobacco pushed *ad nauseam*

[1] *Loc. cit.* p. 159. [2] Hyde Salter, *op. cit.*, p. 235.
[3] *Ibid.*, p. 238.

is said to be the only thing that gives any relief.[1] Trousseau, also, was benefited by a few whiffs of a cigar.[2] But the drawback is, that those who are ready to submit to the unpleasant symptoms that tobacco and stramonium produce, soon become habituated to these remedies and lose the susceptibility to their influence. Théry reports the case of a patient who, after long-continued use of stramonium, suffered from the symptoms of poisoning produced by it.[3]

The irritation of the conjunctival and nasal mucous membranes in cases of hay-fever is best relieved by frequently bathing them with tepid water.[4] The fits of sneezing are benefited by injections of water, or by the use of the nasal douche.

d. Obstruction.—The principles of treatment just alluded to are applicable also to those cases in which the dyspnœa is due to obstruction of the bronchi by viscid sputa. The plugs of mucus are, practically, foreign bodies, the displacement of which, so as to allow the access of air to the lungs, is alone capable of procuring relief. Inhalations of salt[5] or of lime-water are at times beneficial. The viscid mucus, also, is softened by inhalations of chloride of ammonium. Trousseau relates the case of a captain who, while

[1] Hyde Salter, *op. cit.*, p. 189. [2] *Loc. cit.*, p. 469.

[3] *Op. cit.*, p. 383.

[4] *Phöbus*, *op. cit.*, p. 231; W. C. Roberts, *New York Med. Gaz.*, October 8 and December 10, 1870.

his ship was carrying guano, was always free from asthma. The fumes of burning nitre paper are occasionally useful; they act by the ammonia which is formed in the process of combustion. Viaud found among them nitrous oxide and an ammoniacal compound resembling aniline;[1] but, according to the analyses of Eulenberg,[2] the fumes consist of cyanogen, of cyanide of potassium, and of ammonia.

Here, again, the dyspnœa interferes with efficient inhalations, and it is necessary, at times, to adopt such measures as assist expectoration by an increased exudation of fluid from the glands and the vessels of the bronchial mucous membrane. Iodide of potassium, in large doses and strongly diluted, produces the desired bronchorrhœa, and its exhibition is indicated in the absence of an acute inflammatory process. Inhalations of oil of turpentine, which stimulates the mucous glands, contribute to the same end. In the cases in which the rhonchi, by their changes of site, show the mobility of the plugs, emetics are useful, since the compression of the thorax in the act of vomiting serves to remove the bronchial obstruction. Subcutaneous injections of apomorphia afford speedy relief, and, in the presence of cyanosis, they are preferable to the internal administration of drugs of similar kind. Gerhadt[3] recommends the manual compression of the bases of the lungs, so

[1] Germain Sée, *loc. cit.*, p. 711. [2] *Op. cit.*, p. 514.

[3] "Die Behandlung des Lungenemphysems durch mechanische Beförderung der Expiration."—*Berlin. Klin. Wochens.*, No. 3, 1873.

as to assist expiration. In this way, the mucous plugs may be easily displaced, and the dyspnœa may be greatly diminished. The compression should be gentle and be made at the end of each expiration, but it should not be continued too long, lest it produce bronchial hæmorrhage.

e. Œdema.—In cases of embolism of the pulmonary artery, stimulants may be required if the energy of the heart is flagging. As a rule, however, the cardiac muscle is quite equal to the task of restoring the circulation of the obstructed territory ; for, frequent as is the plugging of the smaller branches of the pulmonary artery, it seldom or never proves fatal, notwithstanding the treatment is, on account of the imperfect diagnosis, often unsuitable. In cases of œdema of the lungs, Traube recommends large mustard poultices to the chest,[1] and acetate of lead in one-grain doses every half hour. Stimulants, however, should be liberally given if the cardiac action show signs of failure.

Nitrite of amyl, which paralyses the cutaneous vessels, and in small doses stimulates respiration, may be cautiously tried in cases of pulmonary œdema. The inhalation of a few drops of it has been known promptly to relieve the dyspnœal attack.[2] Pick[3] relates cases in which the asthmatic seizures that

[1] "Charité Annalen."—*Jahrg.*, 1874, p. 269.

[2] Talford Jones, *Practitioner*, Oct. 1871, p. 213; Kitchen, *Americ. Journ. of Insanity*, October 1873.

[3] *Das Amylnitrit.* Berlin, 1877, p. 60, *et seq.*

resisted every other remedy could be mitigated only by that substance.

2. *Symptomatic indication.*—There are, as a rule, but few cases in which the diagnosis remains doubtful, or in which the causal indication cannot be successfully met. Under these circumstances, if the paroxysm be protracted and severe, the relief of the *symptom dyspnœa* becomes the paramount object of treatment. That object, however, can be attained only by suppressing the irritability of the nervous system ; and, in attempting to do so, it is well to remember that, desirable as a speedy relief is, it is yet not purchased without, at the same time, incurring considerable risk. It is superfluous to trace the stages by which asthmatics become gradually habituated to the use of such remedies as temporarily alleviate their sufferings. It is sufficient to say that chloroform and morphia in particular are often continued much more on account of their exhilarating effect than for any other purpose. Instances are not wanting in which such purely symptomatic treatment has led to consequences more painful and also more serious than the disease itself is capable of producing.[1]

Among the remedies most frequently used for the purpose of reducing the irritability of the respiratory centre are :—

a. Chloroform.—The inhalation of a stimulating dose of chloroform occasionally arrests the paroxysm ;

[1] Conf., Salter, *op. cit.*, p. 221-2; Trousseau, *op. cit.*, t. ii, p. 478.

whereas, if the drug be pushed to narcotism, the dyspnœa generally returns with the restoration of consciousness. The patient should, for obvious reasons, never administer the chloroform himself.

b. Chloral.—Chloral in small doses is often of uncertain operation in asthma, and in large doses it is dangerous on account of its paralysing influence upon the centre of respiration. Its exhibition, therefore, requires some caution, and it is contraindicated by cardiac affections, especially by dilatation that accompanies mitral regurgitant disease.

c. Morphia.—Morphia is best given in subcutaneous injections. Vibert[1] recommends the use of small quantities at a time, from one-thirtieth to one-twentieth of a grain, and a repetition of the operation in from ten to twenty minutes, until the pupil commences to contract. The dyspnœa, however, generally ceases after one or two injections. The abdomen, he thinks, is the place where the introduction of the canula is least painful. It is not superfluous, perhaps, to add that such injection should be looked upon as a surgical operation, and that the patient should not be entrusted with the syringe. In pusillanimous patients morphia may be given in a suppository. Opium, in conjunction with such stimulants as ether or ammonia, also procures temporary relief.

[1] Virchow's *Jahresbericht*, 1875. Bd. i, p. 508 *seq.*

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